A STUDY ON THE EFFECTIVENESS OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS

Dissertation submitted in partial fulfillment of regulation for

The Award of M.S. Degree in General Surgery (Branch I)

THE TAMILNADU

DR.M.G.R MEDICAL UNIVERSITY

CHENNAI –APRIL, 2014

COIMBATORE MEDICAL COLLEGE HOSPITAL

CERTIFICATE

This is to certify that this is the bonafide dissertation done by **Dr. Akshay Omkumar** from September 2012 to November 2013 and submitted in partial fulfilment of the requirements for the Degree of M**. S. General Surgery**, Branch I of The Tamilnadu Dr .M .G .R. Medical University, Chennai.

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ACKNOWLEDGEMENT

I thank the Dean **Dr.R.VIMALA M.D.** Coimbatore Medical College and Hospital, Coimbatore for having allowed me to do this study in this Hospital.

I am immensely thankful to Prof. **Dr.V. ELANGO** Professor and Head of the Department of Surgery for his excellent guidance in conducting this study.

I take this opportunity to express my deep gratitude to my beloved chief, Prof. **Dr. G. RAVINDRAN M.S,** Professor of Surgery whose guidance and criticism which was helped me through the entire course of this study.

I would also like to thank all other Unit Chiefs Prof. **Dr. P. Swaminathan M.S**,Prof. **Dr. D.N. Renganathan M.S**, Prof. **Dr.S .Natarajan .M.S** and Prof.**Dr.S.Saradha M.S** for supporting me throughout this study.

I would also like to convey my sincere thanks to our unit Assistant Professors **Dr. S.Muthulakshmi M.S** and **Dr.S.Karthikeyan M.S** for their timely guidance. I would also like to the thank Prof.**Dr.N. Murali M.D ,** Professor and Head of the Department of Radiology for providing timely help with the Doppler imaging studies.

I thank all the patients whose patience and co-operation has made this work possible.

DECLARATION

I hereby declare that the dissertation entitled "**A STUDY ON THE EFFECTIVENESS OF WELLS CRITERIA FOR DIAGNOSING DEEP VEIN THROMBOSIS"** was done by me at Coimbatore Medical College

Hospital Coimbatore – 641018 during the period of my post graduate study for M.S. Degree Branch-1 (General Surgery) from 2011 to 2014. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University

in partial fulfillment of the University regulations for award of M.S., Degree in General Surgery.

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E-mail	akshayomkumar@gmail.com
Submission time	27-Nov-2013 11:59PM
Total words	12478

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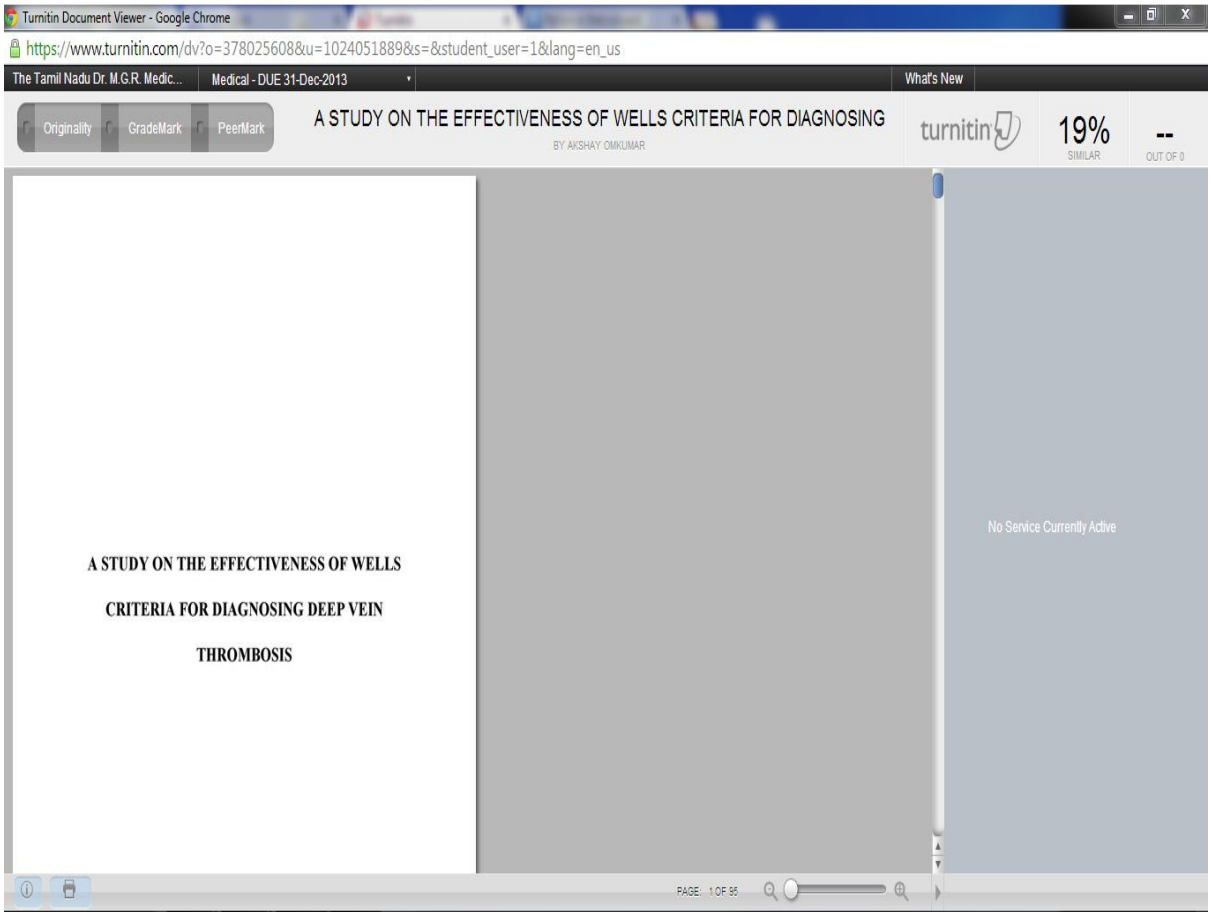

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Keywords: Deep vein thrombosis, Wells criteria , Doppler ultrasound

Abstract:

A clinical criteria devised by Wells and co-workers to diagnose deep vein thrombosis namely “Wells Criteria” has been tested in our study. This criteria has been tested in various health care settings in New Zealand and has helped to reduce the dependence on radiological investigations .

Aim of the study:

To test the effectiveness of Wells criteria for diagnosing deep vein thrombosis.

To find the associated co-morbidities causing deep vein thrombosis.

Methods:

In our study the Wells criteria was tested using venous doppler ultrasound to confirm any case of deep vein thrombosis as suspected by Wells criteria. Time duration of the study was from September 2012 to November 2013.The study group included 50 cases of deep vein thrombosis as diagnosed by Wells criteria. Along with this the associated co morbid conditions in each case was also documented.

Results:

Fifty cases of suspected deep vein thrombosis as per Wells Criteria admitted to **Coimbatore Medical College Hospital** were subjected to the confirmatory test of

doppler venous ultrasound out of which 46 were proven to have deep vein thrombosis which amounted to 92% of cases. Higher the score more was the probability of having deep vein thrombosis.

Out of the associated co morbidities analyzed surgery was found to be leading factor responsible which was amounting to 43% of cases analyzed. In this group alone 90% of the surgical cases were post caesarean section which signified the prothrombotic status of pregnant females who were in the age group of 20 and 30 years .

There was also a relation between blood group and deep vein thrombosis in which 47.8% of cases had A positive blood group indicating a higher level of Von Willebrand factor, possibly a mutant variant.

Conclusion:

We can conclude that the Wells criteria is a very efficient indicator to diagnose deep vein thrombosis in our setup and to classify patients into various risk groups. Thus it would help to reduce delays in diagnosis and aids the clinician to start early treatment for deep vein thrombosis.

INTRODUCTION

One of the most important problems in the diseases of the peripheral veins is concerned with intraluminal clotting. In 1929, Dencke pointed out that soft friable thrombi could develop in the veins of the calf and the foot purely as a result of stasis. Oschner and Debakey distinguished thrombophlebitis (inflammatory) from phlebothrombosis(non-inflammatory).

Phlebothrombosis is a disease which remains symptom free till complications like pulmonary embolism becomes obvious. It was considered that the efficient return of blood from the veins of the extremities depends on six factors:

1)The circulation time-directly related to the functional efficiency of the Heart.

2)The compressive action of muscle contraction on veins.

3)The absence or elimination of mechanical obstruction.

4)The maintenance of normal negative pressure within the abdomen and thorax.

5)The gravity.

6)An efficient /normal peripheral arterial flow.

The disturbance of any one or a combination of these factors may result in sufficient stasis to produce phlebothrombosis. Phlebothrombosis of deep veins is clinically silent and manifests only as its complications.

Early recognition of the process of venous phlebothrombosis, its pathophysiology and availability of Doppler study has remarkably reduced the incidence of Pulmonary emboli and its consequences by instituting early management.

The human vein is an organ that has been given importance since ancient times. Historians, religious clerics, physicians, politicians even Presidents have quoted about veins during various phases of time.

Rabindranath Tagore, had said "The same stream of life that runs through my veins night and day runs through the world and dances in rhythmic measures."

Among the Greeks it was claimed that Asclepius the God Of Medicine drew blood from the Veins on the right side of Medusa's head to bring forward untold healing powers , even giving life back to the dead.

Sir William Osler (1849-1919) ,a Canadian physician had once quoted "Varicose veins are due to improper selection of grandparents."

As per traditional belief the vein was even believed to be a vein that ran straight from the heart to the fourth finger of the left hand. This was why the fourth finger was initially known as the ring finger where engagement rings are worn.

However all said and done man remains unwary and takes for granted the many magical functions that our veins do quietly in the background until they start malfunctioning.

Deep vein thrombosis (DVT) is a preventable condition that causes significant morbidity and mortality. Statistics show that complications from DVT kills more people than breast cancer and AIDS combined.

The definition of DVT as given by national institute of health -- “A condition in which blood clot forms in a vein that is deep inside the body” .DVT is most commonly known to occur in the lower leg and thigh and if it get dislodged can reach the lungs causing pulmonary embolism (PE) which can be potentially fatal. An equally fatal condition can occur when blood clot reaches the brain causing a cerebral stroke.

Factors favoring the formation of DVT are known as Virchow’s triad—1) venous stasis , 2)state of hypercoagulability and 3) damage to endothelial wall.

Other factors associated with DVT include traditional cardiovascular risk factors namely hypertension, obesity and diabetes.

Some of the contributing factors for DVT adults are cancer, old age, prolonged period of immobilization ,paralysis or stroke, previous history of venous thromboembolism ,congestive cardiac failure, pregnancy or puerperium, hormonal treatment, dehydration, varicose veins, long air travel, inflammatory bowel disease, rheumatoid disease and nephrotic syndrome.

Even though there is so much of information in the surgical community regarding DVT and its dreaded complications , information as to when to start prophylaxis is always a matter of debate especially in post operative patients due to the fear of profuse bleeding during the post operative period.

Current contraindications against initiation of prophylaxis against DVT (with low molecular weight heparin(LMWH)) include intracranial bleed, internal bleeding from the raw wounds and operated sites bleed , spinal bleeding following spinal anesthesia and spinal injury due to hematoma.

The DVT results in the patient suffering which is significantly higher in the post op period or during period of hospitalization. This causes significant raise in the expense to the patient due to prolonged period of stay in the hospital and also ends in wastage of precious hospital resources and manpower.

The clinical diagnosis of DVT is not only challenging but may mimic other conditions as well, thereby making it risky to start empirical therapy with anticoagulants. Advanced imaging facilities such as Doppler venous ultrasound may not be always available in peripheral hospitals. Due to delay in the diagnosis, the treatment also gets delayed resulting in wasting precious time. There are many instances in which clinical assessment with Homan's and Mose's sign have yielded false positives and assessment based on these clinical signs alone may be inadequate.

A clinical scoring system has been developed namely Wells Score which takes into account various aspects in the history as well as various clinical signs which can help the clinician to arrive at a diagnosis of DVT. This helps to save time and money that is wasted in doing many unnecessary investigations .

The aim of my study is to test the application of the Wells Score in our clinical set up and to see how effectively we can diagnose DVT .

REVIEW OF LITERATURE

The Vein:

Veins just like their counterparts i.e. arteries are classified into large ,medium and small with no clear cut demarcations as they blend into one another. They are made up of three layers namely tunica intima, tunica media and tunica adventitia. But these three layers are not as well defined as in arteries. In large veins the adventitia is much larger than the media whereas in large arteries the tunica media is much thicker. Due to this very reason veins are not able to retain their shape. The amount of elastic tissue in veins is also much less compared to arteries. Even in some larger veins the internal elastic membrane may not be well developed or even absent. Valves are an important part of veins which serve to prevent backflow especially from the lower extremities.

Large Veins:

The vena cava along with the portal veins including their tributaries constitute the largest vein of the body. Here the tunica intima is constituted by an endothelial lining along with its basement membrane and some sub endothelial connective tissue with some smooth muscle cells. This blends with the tunica media which is relatively thinner and may have some collagen fibers. Next there

is a thick layer of tunica adventitia which is the distinguishing feature. It is predominantly made up of collagen fibers, some elastic fibers and fibroblasts along with prominent longitudinally arranged smooth muscle bundles.

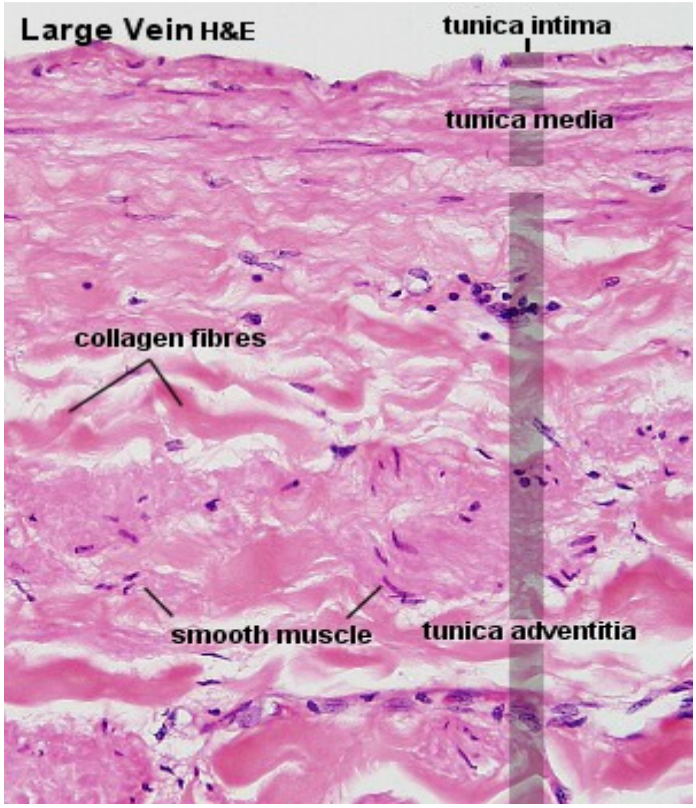


Fig.1

Medium Veins

The tunica consists mainly of endothelium along with a thin sub endothelial layer with smooth muscle among other connective tissues. Internal elastic membrane may or may not be present.

The tunica adventitia remains thicker than the tunica media but is still much less compared to arteries. Medium veins are known to contain valves.

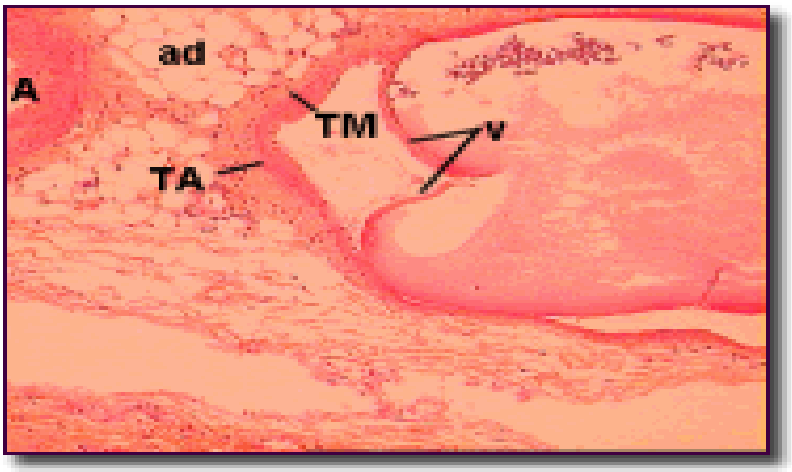


Fig. 19 Medium vein with valve.

Fig.2

Valves can be seen as one or two bands of tissue within the lumen of a vein. Each one of these bands are formed when two layers of tunica intima oppose.

The origin of the bands may from the inner aspect of the venous wall or they can also have separate origins. The manner in which the tissue bands fold forming the valves is variable.

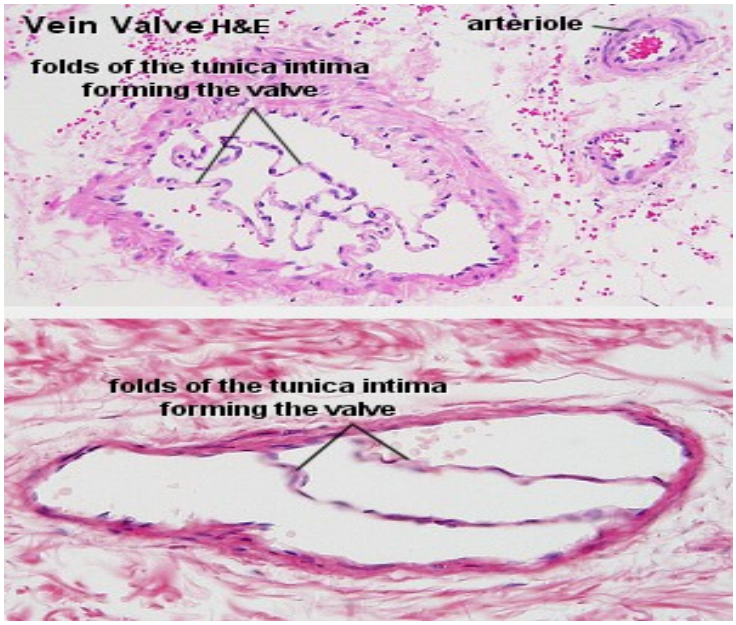


Fig. 3

Small Veins or Venules**:**

Even though various types of venules have been described they cannot be distinguished from each other by looking under a light microscope. Post capillary venules are those that collect blood from the capillaries .They have only an endothelial lining it tunica intima. They are surrounded by undifferentiated mesenchymal cells known as pericytes. White blood cells leave the blood and enter the tissue at the level of post capillary venules .Thus vasoactive agents like serotonin and histamine act on the endothelial layer of post capillary venules and cause extravasation of fluid and RBC's during any inflammatory or allergic reaction.

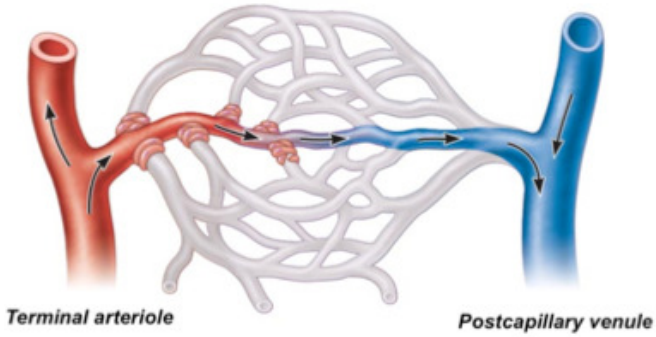


Fig.4

Collecting venules are known to have a thin adventitia along with the pericytes that surround the intima. The adventitia is made up of longitudinally arranged collagen bundles with few elastin fibres.

Muscular venules have around the intima,1-3 layers of smooth muscle and along with an adventitia as said above.

The Venous System

The peripheral venous system acts both like a reservoir to contain extra blood and as a conduit to channel blood from the periphery all the way to the heart and lungs. Unlike the arteries, which have three well-defined layers, majority of the veins are composed of a single tissue layer.

The largest veins with a wide lumen possess internal elastic membranes and at most this layer is thin and not evenly distributed, thus providing little support against high internal pressures.

The proper functioning of the venous system is based upon a complex framework of valves and pumps which are individually frail and thus liable to malfunction, in spite of this the system put together is able to function remarkably well under extremely demanding conditions.

The cardiac output which can come up to 5–10 L/min is received by end-capillary venules which is eventually delivered back to the heart and lungs.

A major portion of this volume flows into the peripheral venous system of the extremities, where it is received and transported against a reverse pressure gradient, following which it is channeled upwards opposite to the direction of gravity and against irregular thoracoabdominal pressures.

This flow is sometimes boosted up by other supportive back pressures such as the increased right atrial pressures of congestive heart failure.

There is no motive force involved which causes such efficient venous return. If all these factors are considered and put together the very mode of functioning of the venous system almost feels magical.

The veins of the lower extremity that are primarily involved in collecting blood are passive, and are thus thin-walled reservoirs that can be easily distended.

Most of these veins are in the suprafascial compartment, which basically consists of only fatty and loose alveolar tissue and thus easily displaced . These veins in the suprafascial compartment can in turn dilate due to the nature of the surrounding tissue to accommodate large volumes of blood with very less increase in the back pressure, so in turn the quantity of blood passing within the venous system at any given point of time will vary by a factor of two or more without any significant alteration of the normal venous function.

The superficial venous system is represented by the veins in the suprafascial compartment. The flow from the collecting veins is via conduit veins whose walls are thicker and thus much less distensible.

These conduit veins are mostly in the subfascial compartment and are thus surrounded by dense connective tissue. As a result it is understandable that these subfascial veins belong to the deep venous system.

Categories of peripheral veins:

1)Suprafacial (superficial)

2)Perforating Veins

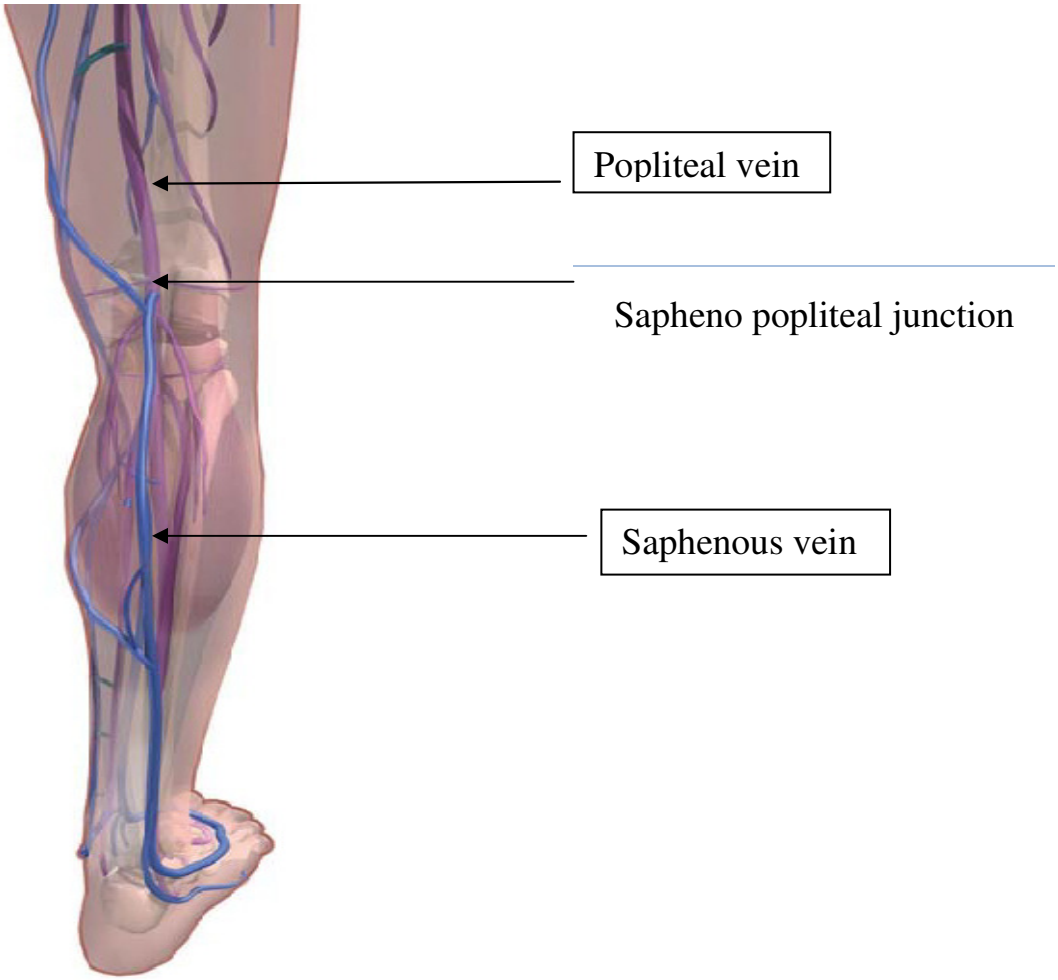
3)Subfascial Veins (Deep)

The Superficial (suprafascial) system of veins:

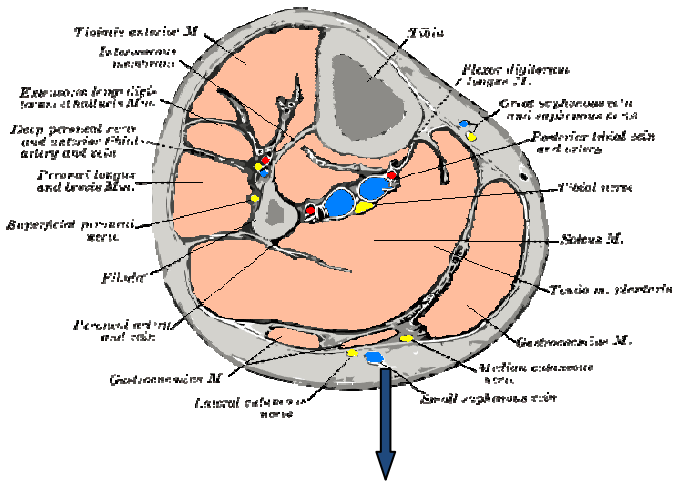
The suprafasical venous system consists of a web like network of veins which are interconnected at various places and intricately arranged and the veins that form this system are mostly unnamed . But there consist of a few superficial veins ,the position of which fairly remain constant and like the deep veins A few larger superficial veins are fairly constant in location; like the deep veins, these superficial veins serve like a passageway to channel blood centrally and finally into the deep venous network. The most important superficial veins of the lower extremity are the short saphenous vein (SSV) that usually runs from the level of ankle to the knee and there is the great saphenous vein which runs from the medial aspect of the ankle to the groin.

The Small Saphenous vein

The origin of the small saphenous vein is in the lateral aspect of the foot. Its course is postero-lateral to the Achilles tendon in the lower aspect of the calf muscle. The small saphenous vein is usually situated directly superficial to the deep fascia over the midline as it reaches the upper calf, where it in turn enters the popliteal space between the two heads of the gastrocnemius muscle.



The small saphenous vein joins the popliteal vein above the knee joint in two-thirds or the cases and in the remaining one-third of cases, it joins with the other veins (mostly the GSV or the deep muscular veins of the thigh). In some of the cases, the SSV may have two or three different termination sites.



Position of small saphenous vein in the leg

The Great Saphenous Vein (GSV)

The GSV arises from the medial aspect of the foot and passes onto the anterior part of the medial malleolus followed by which it, then crosses the medial aspect of tibia posteriorly to rise medially across the knee joint. Above the level of the knee, it continues antero medially, superficial to the deep fascia, and

passes through the foramen ovale joining the common femoral vein at the groin crease at a site termed the saphenofemoral junction (SFJ).

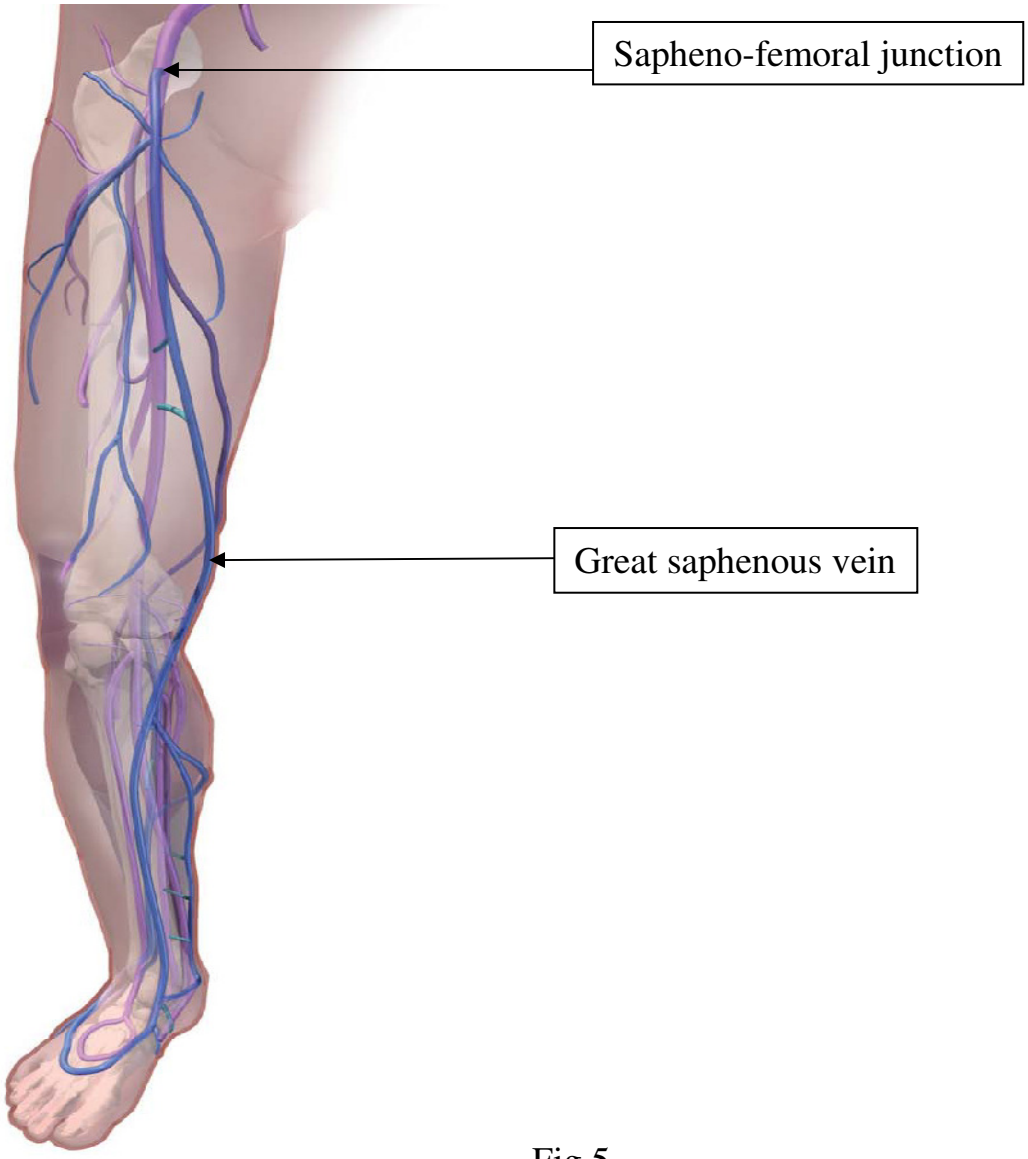


Fig.5

Large tributaries of the GSV are usually mistaken for the main trunk. Majority of patients have at least two major tributaries below the knee ie .the anterior and posterior tributaries, the latter known as the posterior arch vein.

Along with this there are also two above the knee which are the anterior circumflex and posterior circumflex tributaries. They usually drain into the GSV distal to the SFJ; but they are also likely to have a direct connection to the femoral vein. Additionally there are three pelvic veins that commonly drain into the GSV at the SFJ: the superficial inferior epigastric, the superficial external pudendal, and the superficial circumflex iliac veins.

Many cases can also have a duplicated main GSV trunk in the thigh and some may have three or even four veins, known as anterior or posterior accessory veins, which parallel the main GSV trunk and either reconnect with it usually just above or below the knee or traverse more superficially in the distal thigh.

Perforating veins

Most of the veins in the superficial compartment collect and deliver their blood into the great as well as the small saphenous veins, following which these in turn supply most of their blood into the deep venous system via the SFJ and the saphenopopliteal junction (SPJ).

However, there are many alternate channels other than the SPJ and SFJ from the superficial system to the deep system. There are a variable number of perforating veins that connect the superficial veins through various openings in the deep fascia to join directly with the deep veins of the calf or thigh.

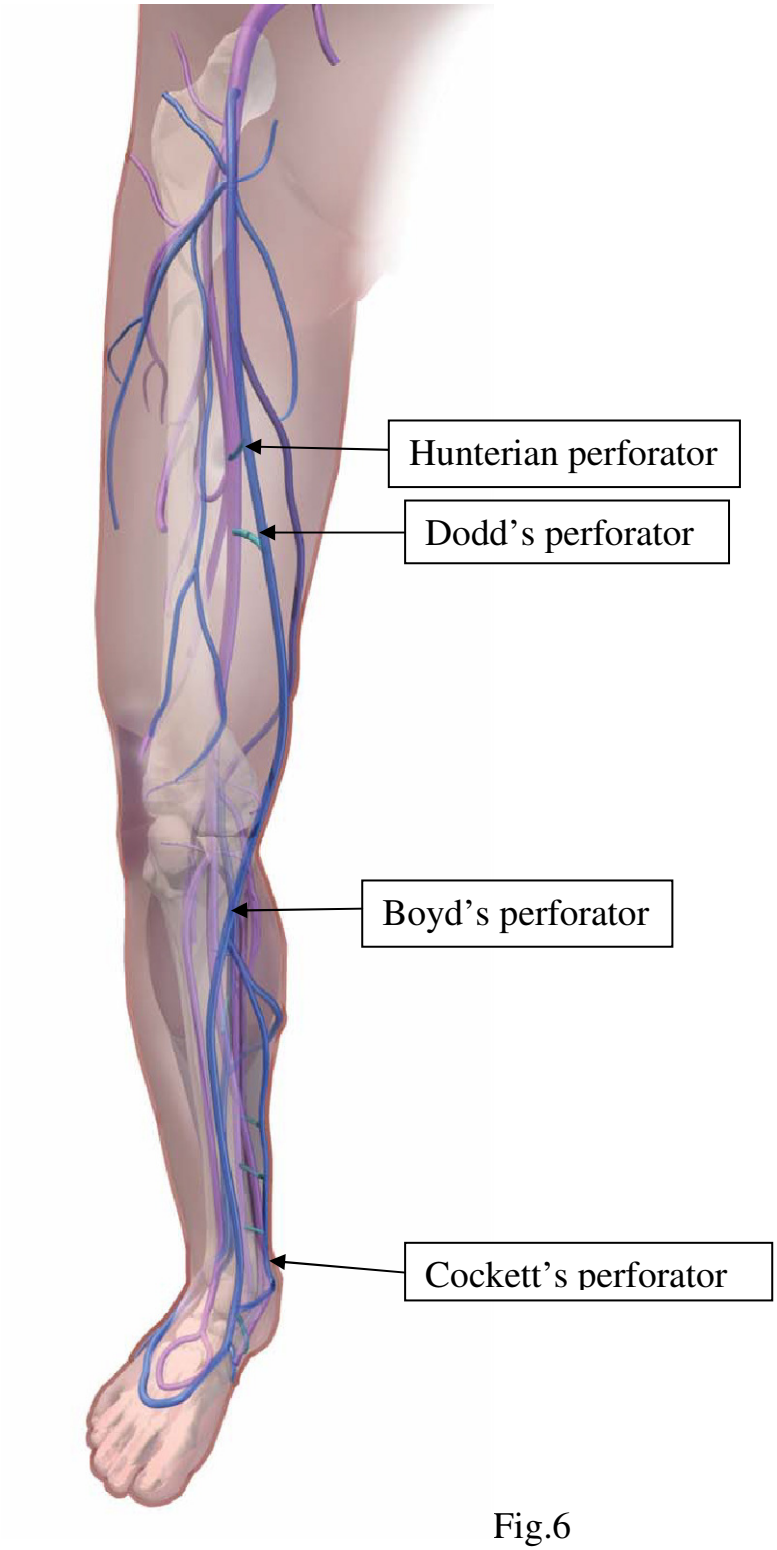


Fig.6

There are various valves in the perforating veins that prevent reflux of blood from the deep veins into the superficial system. There are a few named perforators that are most likely known to be constant in position and are known

only as vague groupings. The old nomenclature includes Hunter's perforator in the mid thigh, Dodd's perforator in the distal thigh, Boyd's perforator at the knee, and Cockett's perforators in the distal medial calf and ankle .

The Deep venous system

The venous blood eventually is channeled into the deep venous system before reaching the right atrium of the heart. In majority of the cases, there are five major named deep venous system veins, of which three are below and two above the level of the knee.

The main deep vein of the leg is known as the popliteal vein (PV) which arises from below the knee and it runs upward and anterior through the adductor canal in the distal aspect of the thigh, where it is known as the femoral vein (FV) for the rest of its course in the thigh.

Although historically called the "superficial femoral vein," this deep vein should be referred to simply as the femoral vein in order to clarify its position within the deep venous system.

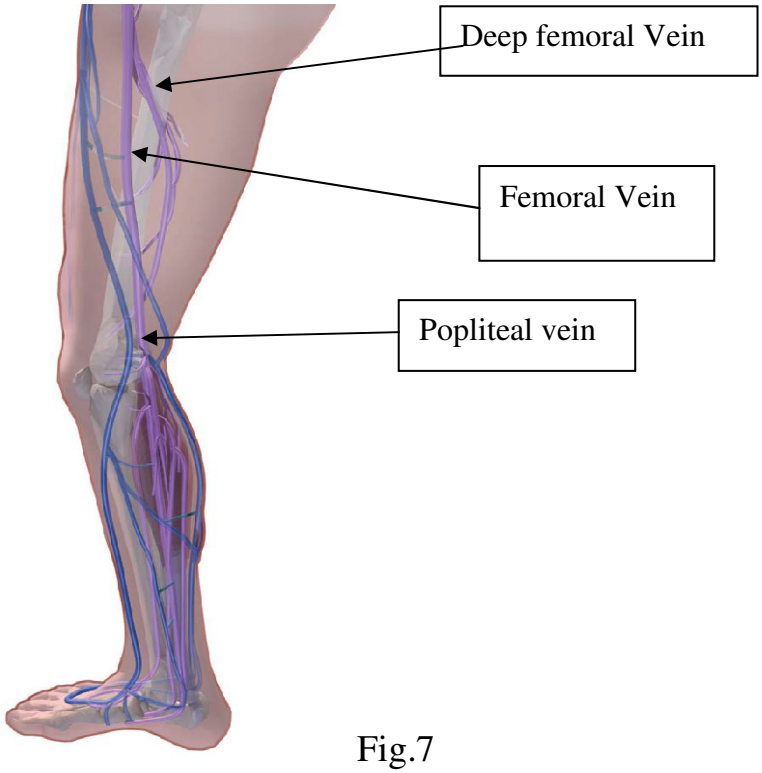


Fig.7

Deep veins of the calf

In the lower leg, there are three pairs of deep veins : the anterior tibial vein (ATV), draining the dorsum of the foot; the posterior tibial vein (PTV), which drains the medial aspect of the foot. Finally there is the peroneal vein, which drains the lateral aspect of the foot.

From above the level of the ankle, the anterior tibial vein courses upward , anterolaterally to the interosseous membrane .The peroneal vein passes superiorly and posteriorly through the calf whereas the posterior tibial vein runs superiorly and posteromedially below the medial edge of the tibia.

Venous sinusoids inside the calf muscle unite to form the intramuscular venous plexi of gastrocnemius and soleus ,which in turn join the peroneal vein at the

level of mid calf. In most individuals, there are six named deep veins below the knee each being a pair of veins which flank an artery of the same name.There are four anterior and four posterior tibial veins fuse with the two peroneal veins to ultimately form a single large popliteal vein just below the knee.

Deep veins of the thigh

The popliteal vein runs behind the knee proximally and then passes anteromedially through the adductor canal in the distal aspect of the thigh, from where it is called the femoral vein. The popliteal vein and the femoral vein are continuation the same vein and thus this is the longest as well as the largest deep vein of the lower extremity.

The deep femoral vein (DFV) originates in the lateral thigh within the deep muscles tributaries is a short and stubby vein and may occasionally communicate with the popliteal vein in up to 10% of patients.

The common femoral vein is formed in the proximal thigh by the union of femoral vein and deep femoral vein. This vein in turn passes upward above the level of the groin crease to become the iliac vein.

Venous valves-

The presence of valves is one of the most important clinical features of veins. These structures are delicate but extremely strong at the same time and lies at

the base part of a segment of vein which expands to form a venous sinus. Due to this arrangement the valves are able to open up properly without coming into contact with the wall. This feature allows rapid closure when flow begins to reverse.

The anterior tibial vein has approximately 9 to 11 valves followed by 9 to 19 in the posterior tibial, peroneal having 7, 1 in the popliteal and 3 valves in the superficial femoral vein . In two thirds of the femoral veins there is a valve present at the upper end at 1 cm proximity to the inguinal ligament. One-fourth of the external iliac veins have valves. No valves are usually present in the common iliac veins.

The number of valves present in the superficial veins are much less, the greater and lesser saphenous veins having around 7 to 9 valves. Venules with a diameter of 0.15 mm have approximately 56 valves.

In most of the areas in the arms and legs valve cusps are oriented in such a manner so as to direct blood flow towards the vena cava and prevent reflux down into the lower extremity.

Usually valves in the perforating veins allow blood to flow from the superficial to the deep venous system , but in the case of the foot the valves also allow flow from the deep to the superficial system.

Venous Circulation [27]

Blood flow that occurs through the blood vessels especially the veins is mainly due to the pumping action of the heart. Flow of blood through the veins is also aided by the rise in the pressure in the thorax during inspiratory phase of respiration , the beating heart as well as compression of the veins by contraction of skeletal muscles which constitutes the muscle pump.

Venous flow and pressure

The normal pressure inside the venules ranges from 12-18 mm Hg. As the caliber of the vein increases the pressure falls steadily to about 5.5 mm Hg inside the great veins that lie outside the thorax.

The intraluminal pressure of the great veins before they enter into the right atrium (central venous pressure) comes to an average of 4.6 mm Hg , but also fluctuates with respiratory movement and heat beat.

Peripheral venous pressure like the pressure in the arteries is altered by gravity. It increases by 0.77 mm Hg for every centimeter below the level of right atrium and decreased by a similar amount for every centimeter above the level of right atrium the pressure is measured.

Thus if the influence of gravity is measured on arterial versus venous pressure there is a greater effect on venous pressure. When blood flow occurs from the venules into the large veins , the average velocity gets increased as the total

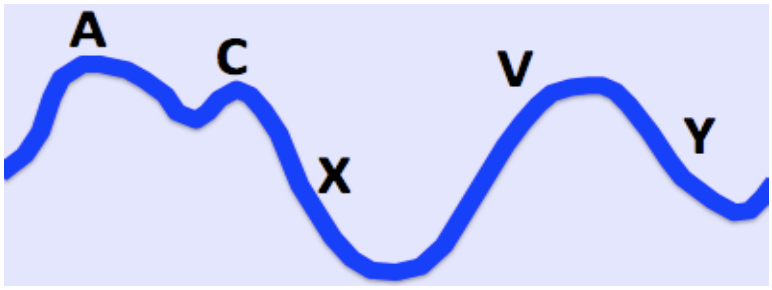
cross-sectional area of the blood vessels decreases .In most of the great veins , the velocity by which the blood flows comes to about one fourth of that in the aorta which averages to 10 cm/s.

Thoracic pump:

While inspiring the fall in intrapleural pressure that occurs is from -2.5 to -6 mm Hg. This fall in pressure is in turn transmitted to the great venous system and to atria in a lesser extent. As a result the fluctuation in central venous pressure ranges from 2mm Hg during inspiration and 6 mm Hg during expiration. This drop in venous pressure during the inspiratory phase aids venous return. Rise in intra abdominal pressure occurs when the diaphragm descends down while inspiring and thus blood is squeezed towards the heart since venous valves prevent backflow into the leg veins.

Effects of heartbeat:

When the variations in atrial pressure are transmitted into the great veins it produces the a,c and v waves of the venous pressure-pulse curve.



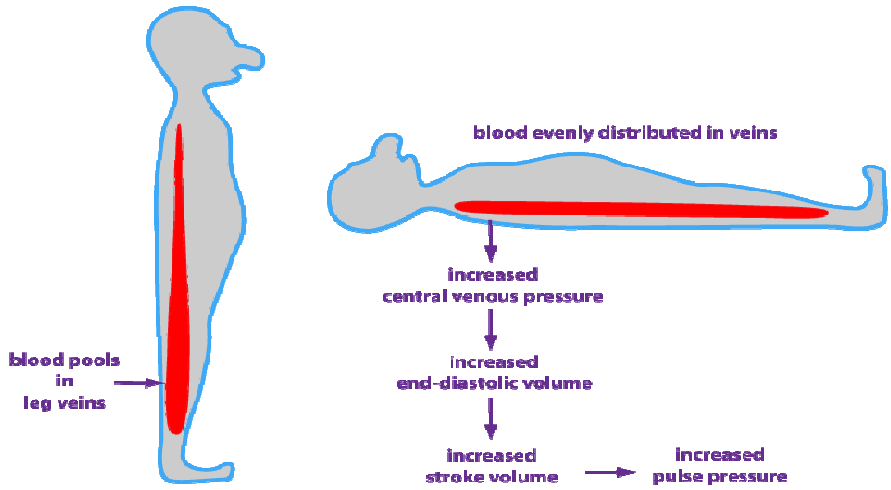
Fig,8

During the ejection phase of ventricular systole the atrial pressure drops sharply because of the pulling down of atrioventricular valves thus increasing the capacity of the atria. As a result of this blood is sucked from the atria from the great veins. This sucking of blood into the atria during systolic phase is mainly responsible for venous return especially when the heart rate is high. Venous flow is pulsatile when in close proximity to the heart. Two periods of peak flow are appreciable especially when the heart rate is slow. One period occurs due to the pulling down of atrio-ventricular valves during ventricular systole and the other during early diastole as part of the rapid filling phase of the ventricle.

Muscle pump

There are various veins in the lower limb which are surrounded by skeletal muscles. When these muscles are put into contraction during any activity this compresses the veins. Veins are also compressed by the pulsations of the adjacent arteries. Blood as a result moves towards the heart due to the valves in the veins that prevent flow in the opposite direction. When standing immobile the full effect of gravity manifests and the venous pressure at the ankle level

becomes 85-90 mm Hg. This results in pooling of blood which reduces venous return as a result of which cardiac output reduces and if this process continues fainting can occur.



.Fig.9

Venous pressure is lowered in the legs to less than 30 mm Hg as a result of contraction of leg muscles in a regular rhythm and thus resulting in propulsion of blood towards the heart. This movement of blood towards the heart is decreased in varicose veins due to the incompetent venous valves. When this condition persists for a long duration the patients develop stasis along with ankle edema.

In spite of the incompetent valves flow of blood can still continue towards the heart due to muscle contractions and also since the resistance offered by the

larger veins which lie in the direction of the heart is less than the resistance offered by the small vessels which lie away from the heart.

Venous pressure in the head

In the standing position the venous pressure in those parts of the body above the level of the heart is decreased by the force of gravity. At a point above which the venous pressure is close to zero, the neck veins collapse. However the dural sinuses cannot collapse since they have rigid walls. Irrespective of the position whether standing or sitting, the pressure is always sub atmospheric. The quantum of negative pressure in the is proportional to the distance vertically above the level of the collapsed neck veins and can be as much as – 10 mm Hg in the superior sagittal sinus.

This is a very important fact which must be remembered by neurosurgeons especially when neurosurgical procedures are performed with the patient in seated position. Air embolism can occur if one of the sinuses gets opened during a procedure.

Measuring Venous Pressure

Central venous pressure is measured in a direct manner by the insertion of a catheter into the great thoracic veins. In majority of the conditions peripheral venous pressure correlates well with central venous pressure. Peripheral venous pressure is measured by measured by inserting a needle attached to a manometer into a vein in the arm.

The peripheral vein should be kept in level with the right atrium which is a point about half the diameter of the chest from the back while being supine. The readings obtained as the dividing by 13.6 (the density of mercury).

As the distance from the heart along the veins increases the peripheral venous pressure also increases in comparison to central venous pressure. The mean pressure that exists in the antecubital vein is normally 7.1 mm Hg, as compared with a mean pressure of 4.6 mm Hg that is seen in the central veins.

An approximate estimate of central venous pressure can be made without any equipment by checking the level to which jugular veins get distended when the subject lies with the head above the heart.

The vertical distance separating the right atrium and the area the vein collapses (which is the place where the pressure inside it is zero) is the level of venous pressure in mm of blood.

Factors that increase CVP include:

- Hypervolemia
- forced exhalation
- Tension pneumothorax
- Heart failure
- Pleural effusion
- Decreased cardiac output
- Cardiac tamponade
- Mechanical ventilation and the application of positive end-expiratory pressure (PEEP)
- Pulmonary Hypertension
- Pulmonary Embolism

Factors that decrease CVP include:

- Hypovolemia
- Deep inhalation
- Distributive shock

Venous dysfunction

Venous dysfunction results due to impairment of venous return for any reason, and can arise from abnormalities within the deep veins, superficial veins, or a combination. It can result from primary muscle pump malfunction, from thrombotic or nonthrombotic venous obstruction or as a result of venous valvular incompetence, which may be limited to a segment or can involve the entire length of the vein. The pressure within the veins of the lower extremity is extremely low immediately after ambulation. The arterial inflow is purely responsible for the inflow to the lower extremity veins. Up to 3-5 minutes of standing is required to fill up the normal venous system. Once entire venous system gets filled, the valves open up and venous pressure increases to a maximum which is exactly equal to the height of a standing column of venous blood starting from the right atrium to the foot. Under this condition there occurs an urge to move the legs, thus resulting in activation of the muscle pumps and thereby emptying the leg veins.

VENOUS DISORDERS

1) VARICOSE VEINS

Varicose veins basically refer to any tortuous, dilated and elongated vein which can be of any caliber.

It includes those conditions causing venous dilatation, the spectrum of which from minor telangiectasia all the way to severely tortuous and dilated varicose veins.

Telangiectasias consist of varicosities which are intradermal small and tend to be cosmetically unappealing but at the same time not symptomatic by themselves in any manner. Reticular veins are dilated veins in the subcutaneous layer that join the tributaries in the main axial or trunk veins.

Trunk veins are those veins with names, such as greater or lesser saphenous veins or their tributaries as well.

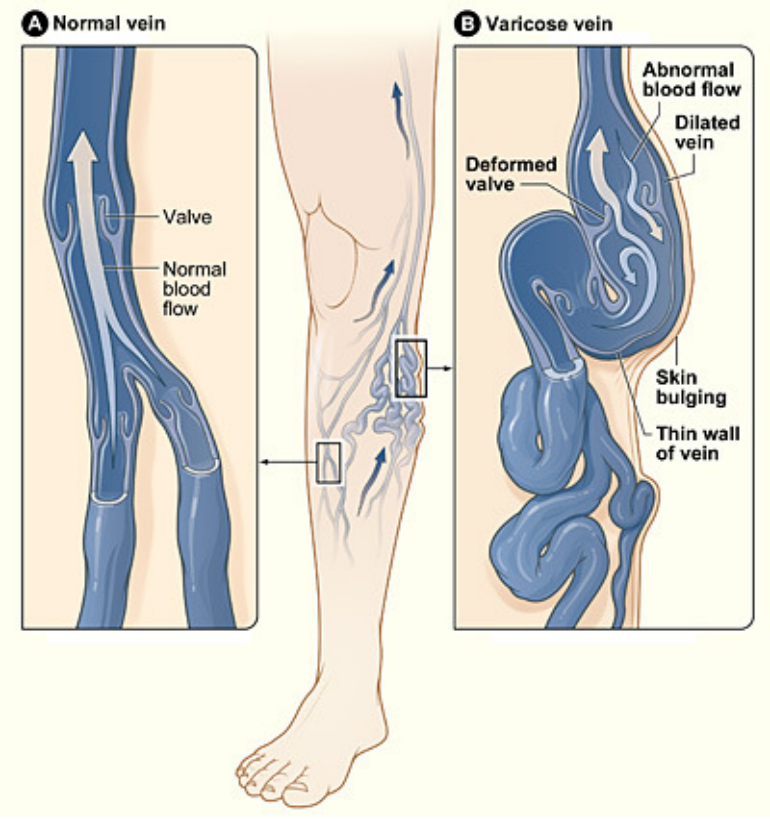


Fig.10

Chronic venous insufficiency towards the end can result in conditions that range from heaviness, aching, pain, and swelling associated with sitting or standing for long durations in the case of varicose veins which are symptomatic. In the other end it can cause severe lipodermatosclerosis along with edema and towards later stages it results in ulceration in those patients with severe chronic venous insufficiency.

Risk factors for varicose veins [4]:

Many risk factors in combination rather than one specific risk factor in general can predict the chances of a patient to develop varicose veins.

Heredity without a doubt has a major role to play in the development of varicose veins.

Valvular insufficiency and dysfunction-

Gravitational force, female sex, hydrostatic force and hydrodynamic forces resulting from muscular contraction.

Hormonal factor- progestrone is responsible for valvular relaxation.

Pathogenesis of varicose veins:

- Pathogenesis of varicose veins is based on the defects in strength and pliability of the walls of veins.

  Defects in the valves of the communicating veins which connect the deep to the superficial compartment may occur .

- According to pressure studies there are two sources of venous hypertension. The first source is gravitational and results from the venous blood flowing in a direction distal to linear axial venous segments situated below. The weight of the blood column from the right atrium is referred to as hydrostatic pressure.

- There is a dynamic component to the second source of venous hypertension. It results from the force of muscular contraction, which is usually maintained within the compartments of the leg. If case of failure of a perforating vein, high pressures which ranges from 150 to 200 mm Hg that develop within intramuscular compartments during exercise are in turn transmitted directly to the superficial venous system. Since the sudden pressure transmitted directly it causes dilation and lengthening of the superficial veins. Further on it leads to increase in the distal valvular incompetence.

- Capillary proliferation is seen in the distal liposclerotic area and a rise in capillary permeability occurs due to the widening of interendothelial cell pores. Transcapillary leakage of fibrinogen occurs which is the principal osmotically active particle.

- Entry of oxygen and nutrients into the surrounding cells is prevented by the extravascular fibrin .However there is very little proof that exists for any actual abnormality for the delivery of oxygen into the tissues.

- Release of proteolytic enzymes from the extravasated lymphocytes is another factor.

Venous thrombosis

This is a condition which can occur either in the superficial or deep venous systems in the leg. Simultaneous involvement of both systems does occur but usually in such cases it begins as a deep venous thrombosis which later on extends to the superficial venous system as well.

In order to get an understanding of venous thrombosis two terms need to be well understood.

Thrombophlebitis refers to the formation of a clot in a vein associated with inflammatory findings such as erythema, tenderness and pain. As a result of the inflammation the blood clot remains firmly adherent to the vessel wall and there are very few chances of it getting dislodged and going to the lungs.

Phlebothrombosis is the formation of a clot in the vein without any signs of inflammation. Here the clot formation is asymptomatic but has more chance of getting dislodged and embolising.

Superficial thrombophlebitis-

Thrombophlebitis usually involves the superficial veins of the lower extremities in comparison to phlebothrombosis which is known to involve the deep veins. Varicose veins are a known pre existing factor in superficial thrombophlebitis of the lower extremities but not an absolute prerequisite. Other contributing factors are trauma to venous wall associated with venous catheters, repeated venous punctures, I.V drug abuse, use of strong IV solutions that produce inflammatory response.

Manifestations of thrombophlebitis-

Dull, aching pain over affected area

Marked redness along vein

Increased warmth over area of inflammation

Palpable cordlike structure

More immediate attention is required if edema, chills, high fever; suggests complications of inflammation.

In cases when thrombophlebitis extends above the level of the knee joint, anticoagulant therapy should be considered if conservative measures fail. The role of anticoagulant treatment is solely for the purpose of preventing thrombembolism and not to treat the primary pathology.

DEEP VENOUS THROMBOSIS [13]

- Acute deep venous thrombosis (DVT) in hospitalized patients is one of the major causes of morbidity and mortality especially in post operative patients.

- Even though its been a hundred and fifty years since Virchow proposed his triad which consists of endothelial injury, venous stasis and hypercoagulable state this still holds true even today . Although the final credit for the pathological triad in DVT has been granted to Rudolph

Virchow much work has been done in the same field by various physicians.

- Wiseman in 1686 had proposed his version of the probable etiology of venous thrombosis which bears a resemblance to Virchows triad.

- He had attributed the cause of venous thrombosis to (1) Coagulation of serum (2) Thickening of blood (3) Decreased venous blood flow by impingement of a tumor or any back pressure built up within the venous system. He also had described the increased incidence of venous thrombosis during pregnancy or any malignant conditions.

- Van Swieten in 1705 was given the credit for describing the increased incidence of clots during puerperium.

- White in 1784 had published that phlegmasia alba dolens is associated with thrombus that develops in the iliac or femoral veins. Phlebitis had been described by Hunter in the same year in his article "Observation of the inflammation of internal coat of veins"

- Virchow upon his appointment in Charite hospital ,Berlin was under the direction of Robert Froriep (1804-1861) who appointed him with investigating the assertion given by French pathologist Cruveilhier who had claimed that phlebitis dominates all pathology. He proceeded with clinical and experimental investigations in the process of pursuing

Cruveilhiers statement. Virchow, in a series of 76 autopsies identified 18 cases of venous plugs out of which there were 11 cases of emboli in the pulmonary arteries. Based on this he coined the term emboli and reasoned that majority of the clots developed in the deep veins of the lower extremities, thereby contradicting the belief that thrombosis occurred in the lungs *de novo* due to inflammation in the veins. He substantiated his necropsy observations in a series of studies where he injected various compounds in to the venous circulation of dogs and by ultimately examining the characteristics of the material that was lodged in the pulmonary vasculature.

- In conditions such as the absence of anticoagulation or even in the presence of inadequacies in anticoagulation the thrombotic process which gets initiated in a venous segment can get propagated and thus involves even more proximal segments in the deep venous system thus causing pain, edema and immobility.
- Pulmonary embolism is one of the most dreaded complications of acute DVT which is a potentially lethal condition.

- Chronic venous insufficiency can occur as a late consequence of DVT especially in the iliofemoral veins causes by valvular dysfunction along with the presence of luminal obstruction.

- Due to these reasons, there needs to be a thorough understanding of the pathophysiology, to standardize the protocols to prevent or reduce the occurrence of DVT, and to thereby institute optimal treatment promptly to such cases. All these steps are critical to reduce the incidence and morbidity caused by this unfortunately common condition.

Etiology –

Soleal sinuses are the most common sites where venous thrombosis gets initiated. Stasis in the sinuses may contribute to activated platelets getting in contact with the endothelial cellular layer as well as procoagulant factors, thereby leading to deep venous thrombosis.

- The Hypercoagulable State

Wiseman (1686) and Andral (1830) had preceded Virchow by suggesting that venous thrombosis was due to the increased coagulating tendency of the blood

Before the 1930's reliable factors that contribute to thrombosis by altering the blood composition were found to be lacking . Although it was known that thrombosis could be precipitated by injection of certain snake venoms only few endogenous factors were known that caused a similar effect.

In 1901 Lotheisen put forward a theory that malignancy, pregnancy and chlorosis along with other factors may affect thrombosis. Following this increase in the levels of fibrinogen, globulin and calcium were also postulated to increase the agglutinability. Later on it was suggested that infection and obesity can potentiate coagulability .

Once any of these conditions favorable for DVT are identified, a treatment regimen which consists of anticoagulation is started for life, unless there are specific contraindications.

Tissue factor may be released after major surgeries into the blood stream in large amounts, the source being damaged tissues. Tissue factor is a very potent procoagulant. Increases in the platelet count, adhesiveness, as well as changes in coagulation cascade, and finally endogenous fibrinolytic activity all are result of physiologic stress such as any major surgery or trauma and all these put together are known to be associated with an increased risk of thrombosis.

Hypercoagulable States:

■ Factor V Leiden mutation

■ Prothrombin gene mutation

■ Protein C deficiency

■ Protein S deficiency

■ Antithrombin III deficiency

■ Antiphospholipid syndrome

Venous Injury

It has been proven that that venous thrombosis occurs usually in veins that are well away from the site of surgery; for example, it is well known fact that patients undergoing total hip replacement frequently develop contralateral lower extremity DVT.

The notion that damage to the intima causes thrombosis known as the “doctrine of Cruveilhier” was known very well prior to Virchow’s research.

Hodgson in 1815 had proposed that trauma to a vein is a predisposing factor to thrombosis. Likewise Davies (1823), Andral (1830) and Lee (1842) had quoted that inflammation of the endothelium was responsible for this phenomenon.

Baumgarten had demonstrated in 1876 that blood trapped in a double ligated vein failed to clot for months together ,but prompt thrombosis occurs when isolated blood with infectious material was inoculated. Barett in 1924 inflicted aseptic trauma to the veins and noted failure of coagulation even in subsequent

experiments even when bacteria was injected at the site of injury. Only when bacteria inoculated threads were placed in the vasculature a thrombus was able to develop. Along with advances in anesthesia thus enabling prolonged surgeries and casualties from many wars the significance of endothelium as a contributor to venous thrombosis was recognized. Intimal injury was thought to be as a result of direct trauma but more importantly due to damage induced by bacterial toxins. Some workers used to even say that thrombosis cannot occur in the absence of infection. Once we can appreciate the complex biology of the endothelium how various factors govern the dynamic relation as to decide the occurrence of thrombosis or thrombolysis.

In case of thrombosis, multiple microtears can be identified within the valve cusps that caused exposure of the subendothelial matrix. It however has not been well understood the exact mechanism which causes injury at a site well away from the operated site and the mediators responsible whether they are cellular or humoral , but the very fact that the injury occurs and occurs reliable is clearly evident from these as well as many other studies.

Venous Stasis

Much prior to Virchow's work, stasis as a significant contributor to venous thrombosis was established by Wiseman's work in 1686 followed by Ballie (1793) ,Davies (1823) , Andral (1830) and finally Bouchut in 1845.

Humphry in 1881 after personally incurring venous thrombosis put forward that clotting of blood occurred as a result of turbulence in blood flow. He said that "eddies" were generated due to the presence of venous valves thus facilitating clotting. It was only 100 years later that the idea of "valve pockets" were popularized. In the 1930's it had come to a consensus that for a thrombus to develop stasis alone was insufficient. In spite of this it was agreed upon that reduced blood flow contributed to thrombosis which is due to the speculation that it results in interaction between the blood components and the vessel wall or by altering the balance of activated clotting as well as inhibiting factors in the blood stream. This hypothesis was additionally supported by the observation of an increased risk of thrombosis in patients paralyzed due to stroke and spinal cord injuries and in patients confined to bed-rest or those who were immobilized. A theory was put forward that in such conditions since the veins were dependent on the pumping action of the adjacent muscles to push back blood to the heart , would develop pooling which would potentiate interaction between the erythrocytes and the vascular endothelium. Recently attention from the media has also revived interest in venous stasis as a contributor to this

condition especially in passengers travelling in trans-oceanic flights and has labeled it as “economy class syndrome”.

Incidence

- Venous thromboembolism occurs for the first time in approximately 100 persons per one lakh individuals. This incidence rate goes up with increasing age with an approximate incidence of 0.5% per 100,000 at the age of eighty
- Out of this group more than two thirds of cases have DVT alone whereas the rest are known to have evidence of pulmonary embolism.
- The rate of recurrence with anticoagulation measures has been found to be 6% to 7% in the ensuing 6 months.
- Chronic Venous Insufficiency (CVI) other than pulmonary embolism which results from DVT causes a significant increase of cost, morbidity, and limitation of lifestyle.
- Regarding the consequences of deep vein thrombosis considering pulmonary embolism and chronic venous insufficiency, is to be prevented, then there must be an optimization of the measures for the prevention, diagnosis, and treatment of DVT.

Clinical Diagnosis

- The diagnosis of DVT requires, to use an overused phrase, a very high index of suspicion.

- **Homans' sign**, which was initially described by American Surgeon John Homans (1877-1954) is basically to pain which occurs in the calf while dorsiflexing the foot. It is most certainly true that though the lack of this sign is not a foolproof indicator of the absence of thrombus is the deep vein, but the once Homans' sign becomes positive it should prompt one to try and confirm the diagnosis of deep venous thrombosis.

- Most certainly, the primary factor responsible for the manifestation of symptoms of deep venous thrombosis is the extent of venous thrombosis seen in the lower limb. For example, most of the calf thrombi may remain asymptomatic unless it propagates proximally.

- In patients with deep venous thrombosis only 40% end up with clinical manifestations.

- Venous thrombosis that involves the major veins such as the iliofemoral venous system will end up with a severely swollen leg associated with pitting edema, blanching and pain, a condition referred to as phlegmasia alba dolens. As the disease progresses, the edema can become so massive that after a point there occurs a compromise in arterial inflow. As a result

a painful blue leg occurs due to this condition, which is called as phlegmasia cerulea dolens.

Phlegmasia Cerulea dolens

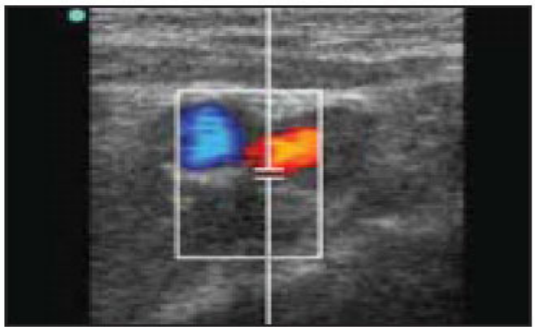
Fig.11

- .This condition, unless flow is restored, will result in venous gangrene . Phlegmasia alba dolens is just a variant of this condition and has associated arterial spasm and the leg will be pale and cool with diminished pulses.

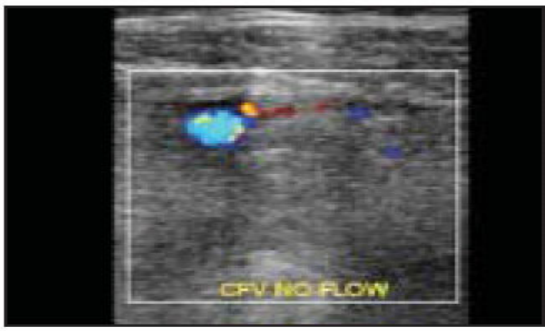
Investigations

Duplex Ultrasound [4]

- It is one of the latest diagnostic test of choice required for the diagnosis of DVT. Duplex ultrasound is a modality which combines real time B-mode ultrasound along with imaging of the color-flow and has thus greatly improved the specificity and sensitivity of ultrasound scanning.
- Since the advent of color flow duplex imaging a thrombus which partially occludes the lumen does not hinder imaging of the blood flow.
- On using the probe to compress the vein a normal vein will be easily compressible whereas when a thrombus is present it offers some resistance to compression.



Color-flow Doppler in normal vessels.



Color-flow Doppler in deep vein thrombosis.

Fig.12

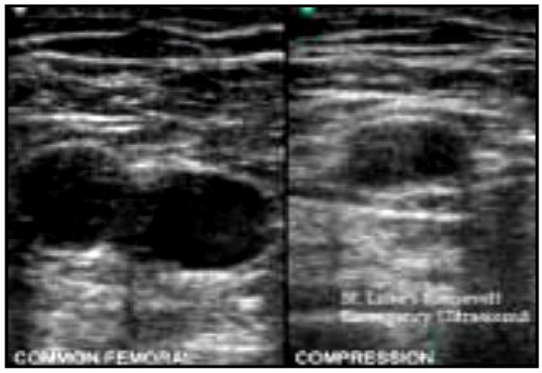
Venous Ultrasonography [4]

This includes examination of the femoral, popliteal, tibial,peroneal, soleal and gastrocnemius muscular veins as routine,plus the iliac veins & IVC where indicated.Patient lies supine – gel on skin

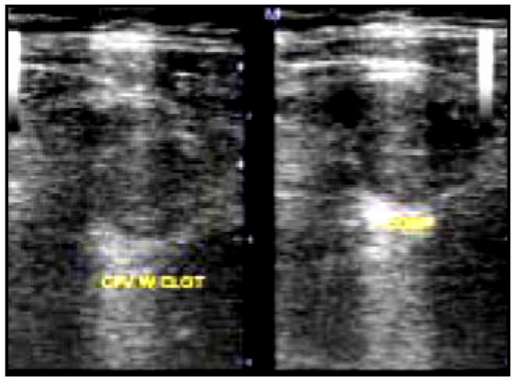
Doppler waveform in groin / common femoral vein (iliacs)

Compression of visualised veins with ultrasound probe

Non compressibility of veins help to identify DVT



Normal compressibility of the common femoral vein.



Abnormal compressibility in deep vein thrombosis.

Fig.13

Technical Limitations – Ultrasound

Large legs, obesity

Cellulitis, ulceration, pain

Adductor canal, iliac veins & IVC

Ruptured Baker's Cyst

Limited mobility eg. post-op

Cardiac failure

Non-occlusive calf thrombi

Magnetic Resonance Venography [4]

- Along with the major advances in technology used in imaging of veins, magnetic resonance venography has come to the frontier of imaging used for proximal venous disease.
- The two factors that limit the widespread its widespread use are the cost factor and patient intolerance due to claustrophobia associated with an MRI machine, but at present with advances in technology this is changing as well.
- It is very a useful test for visualizing the inferior vena cava as well as the iliac veins, both being areas where duplex ultrasound has proven to be inadequate.
- . There are also various situations in which ultrasound cannot be used since it requires direct contact with the limb surface especially in plaster casts.

- There are also various blind spots in ultrasound such as over the gastrocnemius and profunda femoris. It also helps to avoid radation exposure compared to venography.

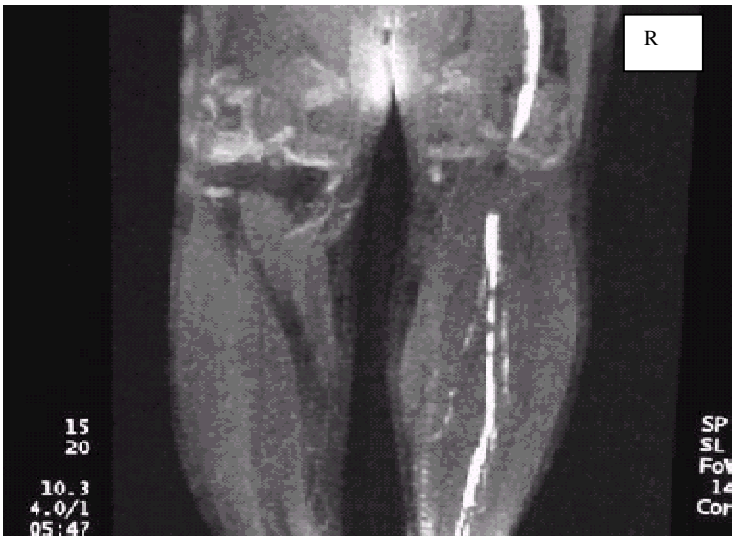


Fig.14

Unilateral DVT in the right limb MR venogram

Venography [4,6,7]

Identifies thrombus in the deep veins mainly of the lower limbs, but also neck, upper limb and abdomen/pelvis.It also helps to identify thrombus in the superficial veins and also any other related pathology. Gold standard scans should include the deep calf veins and extend proximally as required. If

thrombus is present there is abrupt termination of the dye column or filling defects will be visualized in various portions of the venous system.

Fig.15

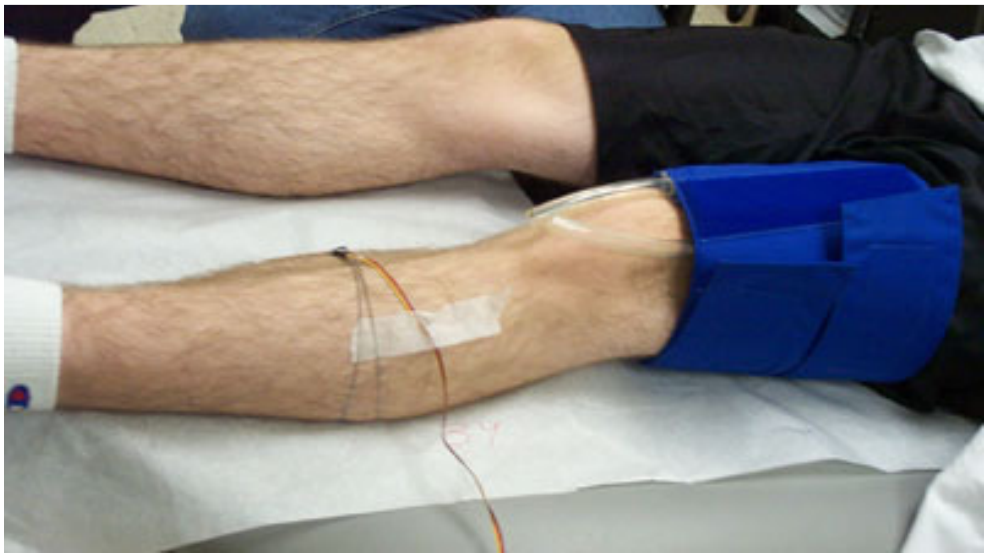
Lower-extremity venogram as shown above depicts acute deep venous thrombosis location being the popliteal vein with enhancement of contrast.

Impedance plethysmography [4]

It is a test which measures volume changes in the extremity. In this procedure changes in electrical resistance of the chest, calf or other regions of the body are measured. Since DVT is associated with edematous changes it is helpful in the diagnosis. A BP cuff is placed in the proximal thigh and inflated to a level between arterial and venous pressures. When this is done a previously positioned calf plethysmograph will record a volume increase.

When the cuff is deflated a reduction in calf volume will occur. The rate at which this volume change occurs reflects the efficiency of venous outflow , thus indirectly indicating the presence or absence of venous thrombosis.

This offers a good advantage in comparison to venography which is invasive and requires a great amount of skill to conduct it and interpret the results effectively.



Impedance plethysmography Fig.16

Other applications of impedance plethysmography:

- Determination of cardiac stroke volume
- Cerebral blood flow measurement
- Determination of body composition
- Intrathoracic fluid volume measurement

Wells Score [1]

Wells score also known as Wells rule was developed by Wells and co-workers .This was developed solely with the intention of reducing the time wasted in unnecessary testing. This test has good sensitivity and specificity in secondary care settings and has helped to reduce the cost of unwanted radiological investigations . The Wells rule simplifies diagnostic assessment by combining various aspects of history, clinical examination and investigations and thus improving clinical accuracy.

This suggests whether a patient is at low, moderate or high risk of having suffered a DVT which may guide subsequent investigation and management .

Wells Clinical Prediction Rule for Deep Venous Thrombosis (DVT)

Clinical feature	Points
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for more than 3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of greater than 3 cm (below tibial tuberosity)	1
Unilateral pitting edema	1
Collateral superficial veins	1
Alternative diagnosis as likely as or more likely than DVT	-2
Total points	

DVT = deep venous thrombosis.

Risk score interpretation (probability of DVT):

- /=3 points: high risk (75%);
- 1 to 2 points: moderate risk (17%);
- 1 point: low risk (3%).

Wells and coworkers have also put forward a similar scoring system for diagnosing pulmonary embolism which has been listed below.

Clinical Characteristic	
Clinical signs of deep vein thrombosis	3
Heart rate >100 beats per minute	1.5
Recent surgery or immobilization (within the last 30 d)	1.5
Alternative diagnosis less likely than pulmonary embolism	3
Hemoptysis	1
Cancer (treated within the last 6 mo)	1
Clinical Probability of Pulmonary Embolism	Score
Low	2
Intermediate	2-6
High	6

Hamilton Score [26]

This is a new score to develop to overcome the limitations of Wells score. The overlapping redundant features, such as lower limb enlargement, calf enlargement, and pitting edema, render the score less accurate in stratification and more cumbersome to calculate. Important risk factors such as prior history

of DVT or pulmonary embolism, pregnancy, and the use of oral contraceptives were not considered when the Wells score was developed.

Hence a new pretest probability scoring system was devised to overcome all these which was Hamilton score.

Plaster immmobilisation of lower limb	2
Active cancer(within 6 months or current)	2
Strong clinical suspicion of DVT	2
Bed rest >3 days or recent surgery within 4 weeks	1
Male sex	1
Calf circumference > 3 cm on affected side (10 cm below tibial tuberosity)	1
Erythema	1

A score of ≤ 2 represents unlikely probability for deep venous thrombosis (DVT); a score of ≥ 3 represents likely probability for DVT

AIM OF THE STUDY

1)To assess the efficacy of Wells criteria for diagnosing deep vein thrombosis.

2)To bring into light the associated co morbid conditions and to find out the most significant one which has got the highest probability for causing DVT.

STUDY DESIGN:

This is a prospective diagnostic validation study of the Wells Rule for DVT in our setup, ultrasound being the gold standard comparison and will be conducted over a duration of 12 months starting from 19.11.2012 to 19.11.2013.

Score for each patient will be calculated based on the parameters given in the table below.

Wells Clinical Prediction Rule for Deep Venous Thrombosis (DVT)

Clinical feature	Points
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Total points	

DVT = deep venous thrombosis.

Risk score interpretation (probability of DVT):

- /=3 points: high risk (75%);
- 1 to 2 points: moderate risk (17%);
- 1 point: low risk (3%).

Fifty patients admitted with a clinical diagnosis of Deep Vein Thrombosis will be considered as the sample size.

Inclusion Criteria:

1)Age greater than 18 yrs

2)Onset of symptoms within 7 days

3)Those consenting to the study

Exclusion Criteria

1)Patients with recurrent DVT

2)Patients with bleeding diathesis

MATERIALS AND METHODS

Fifty patients admitted with a diagnosis of deep vein thrombosis in various departments in Coimbatore Medical College will be studied prospectively between September 2012 and November 2013.

All patients with suspected Deep Vein thrombosis will be assigned a Wells score on admission will be followed up by venous Doppler ultrasound to confirm the diagnosis.

The correlation between the Wells score and Doppler will be checked.

Basic blood investigations such as complete blood counts,blood urea, blood sugar,serum creatinine and electrolytes

along with a chest xray and electrolytes will be sent. A urine routine examination and a chest xray will be taken.

SAFETY CONSIDERATIONS:

In every step of the study patient safety will be given topmost preference. All procedures will be done under strict aseptic precautions and only necessary investigations will be carried out after carefully evaluating the patient.

Any procedure will be done only after getting consent of the patient or the attender.

QUALITY ASSURANCE:

Strict care will be taken to ensure that the study is done in the best quality possible right from patient admission

which includes interaction with the patient,history taking ,clinical examination and while subjecting to the

necessary investigations.

EXPECTED OUTCOME:

This study is basically to test the effectiveness of Wells criteria as a diagnostic tool in Deep Vein Thrombosis.

Wells score application will allow the clinician prepared for the diagnosis even before the confirmation by a Venous

Doppler ultrasound thus saving time in initiating treatment and thereby enabling faster patient recovery .

RESULTS

The Wells score was applied on a total of 50 cases. Out of the total number of cases it was able to diagnose DVT in 92% of the cases ie 46 out of 50 cases. Thus proving to be a very accurate indicator for DVT .

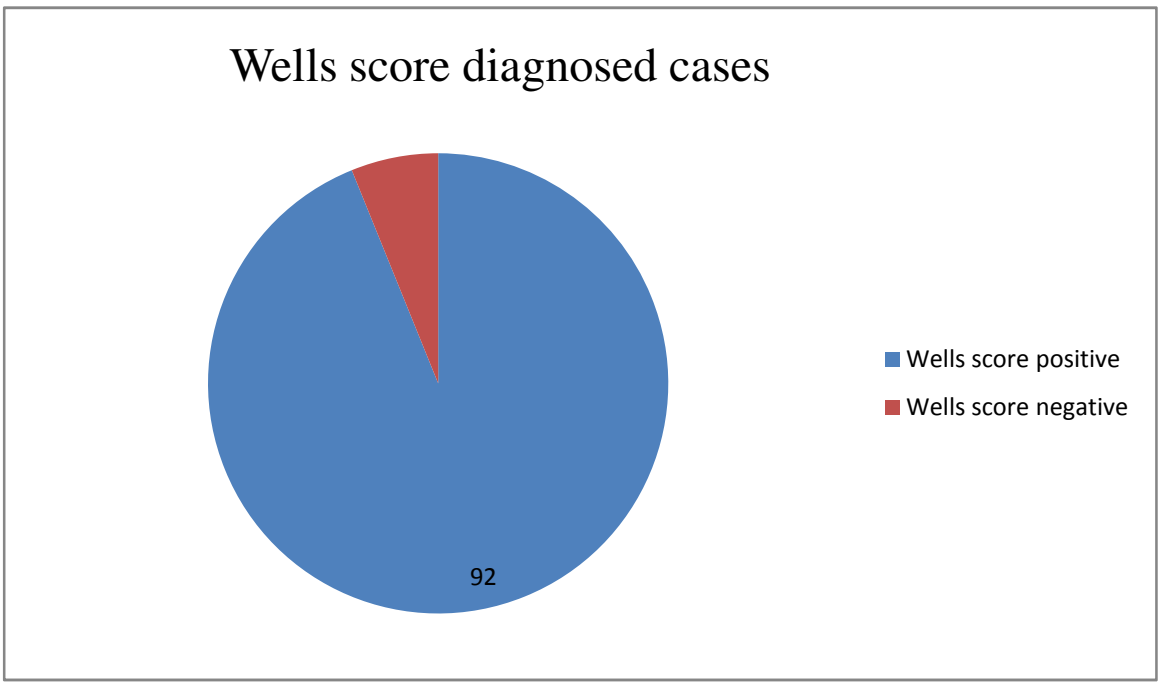

Fig.17

Wells score false positive

On analyzing the Wells score negative cases two of the cases were symptomatic due to acute lymphedema in elderly woman suffering from intra abdominal malignancy and the other two cases were early onset cellulitis.

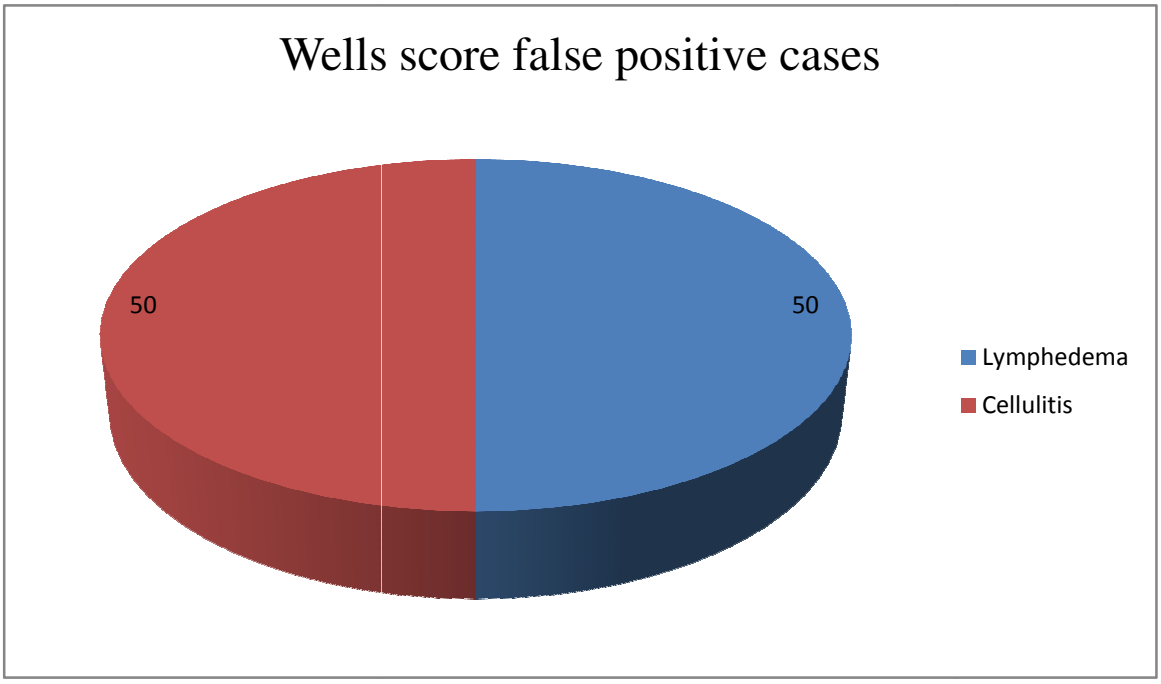

Fig.18

Out of the 4 cases which did not have DVT the highest wells score was 5/8 which included 2 elderly women with intra abdominal malignancy –namely carcinoma of the cervix thus blocking all the lymphatics resulting in tense lymphedema of the leg .The entire leg thus having the appearance of inverted beer bottle appearance similar to acute DVT. Both cases had undergone radiotherapy for cervical cancer a few years ago.

Wells score distribution

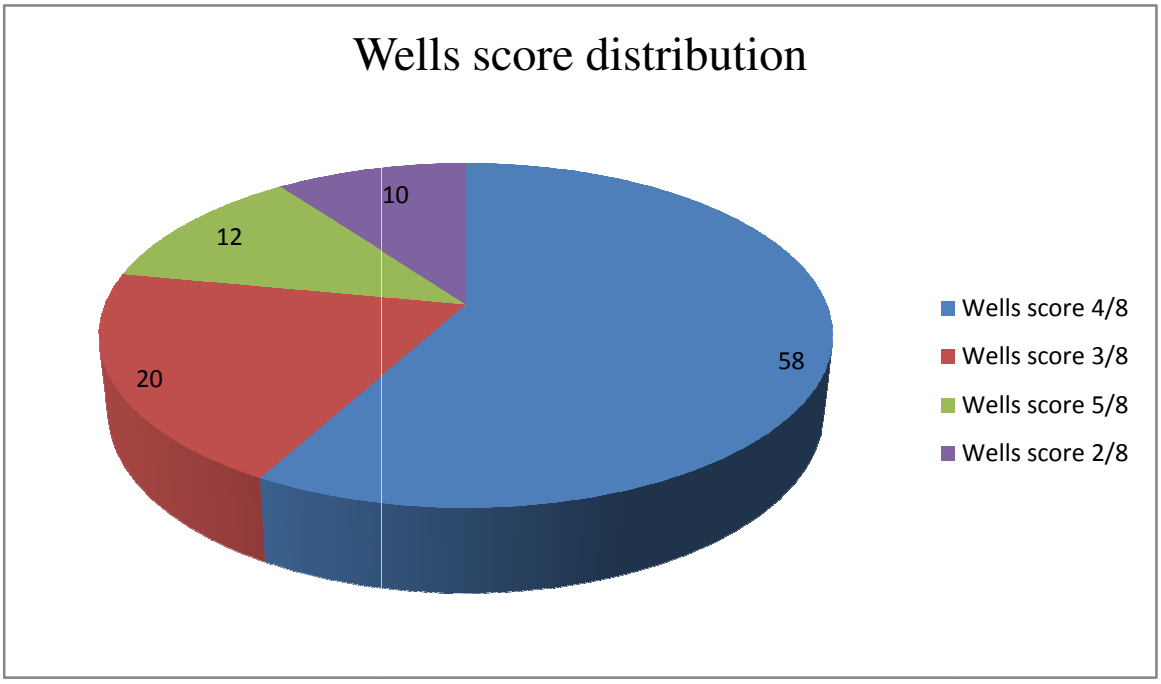

Fig.19

Most of the cases diagnosed with DVT had a Wells score of 4/8 which being above 3 thus indicates a 75% risk of having deep venous thrombosis.29 of the cases had a Wells score of 4/8. Followed by 10 cases which were also in the same risk category with a score of 3/8.

Six cases had the highest score of 5/8 which were also in the highest risk category out of which 2 did not have DVT and were having lymphedema.

The other 2 cases were having the lowest score of 2/8 which put them in the moderate risk category for having DVT which was a risk of 17%.

Blood group distribution in DVT cases

On observing the distribution of blood groups in the entire case list of DVT confirmed cases 22 of the 46 confirmed cases of DVT ie 47.82 % had blood group A positive. This was followed by 19 cases which amounted to 41.30 % which had blood group B positive, the rest were AB positive ie 3 amounting to 6.52 % and finally O positive which consisted of 2 cases amounting to 4.34 % of the cases.

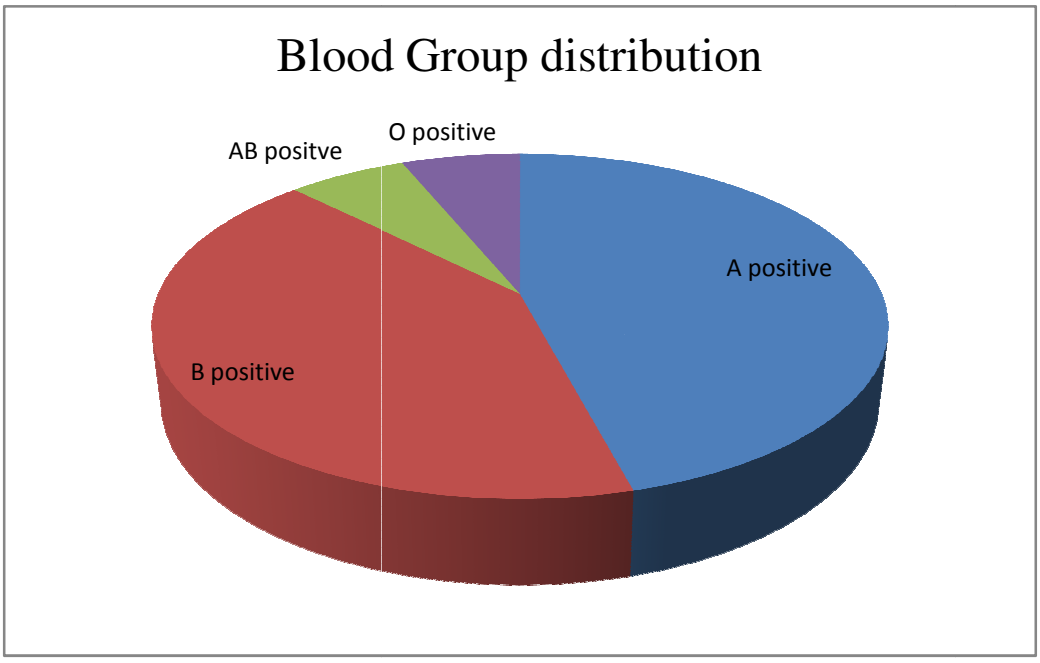

Fig.20

This in turn compliments various studies which suggest a higher rate of deep vein thrombosis in individuals with blood group A .

Sex Distribution of DVT cases

On taking into account the sex distribution of cases of deep vein thrombosis .it can be seen that the number of DVT cases are much higher in women on our hospital due to the high incidence of DVT in post partum females.

This is almost double the ratio of males affected by DVT . 29 females out of the total of 46 were affected. This constituted 63% of the total number confirmed cases of deep vein thrombosis. From this ratio we can understand that hormones namely estrogen and progesterone do have an important role in the causation of deep vein thrombosis as evident from the high number of females affected with DVT.

The risk of deep vein thrombosis also increases in young females who are ooral contraceptives and as well as post menopausal women who are on hormone replacement therapy. [4]

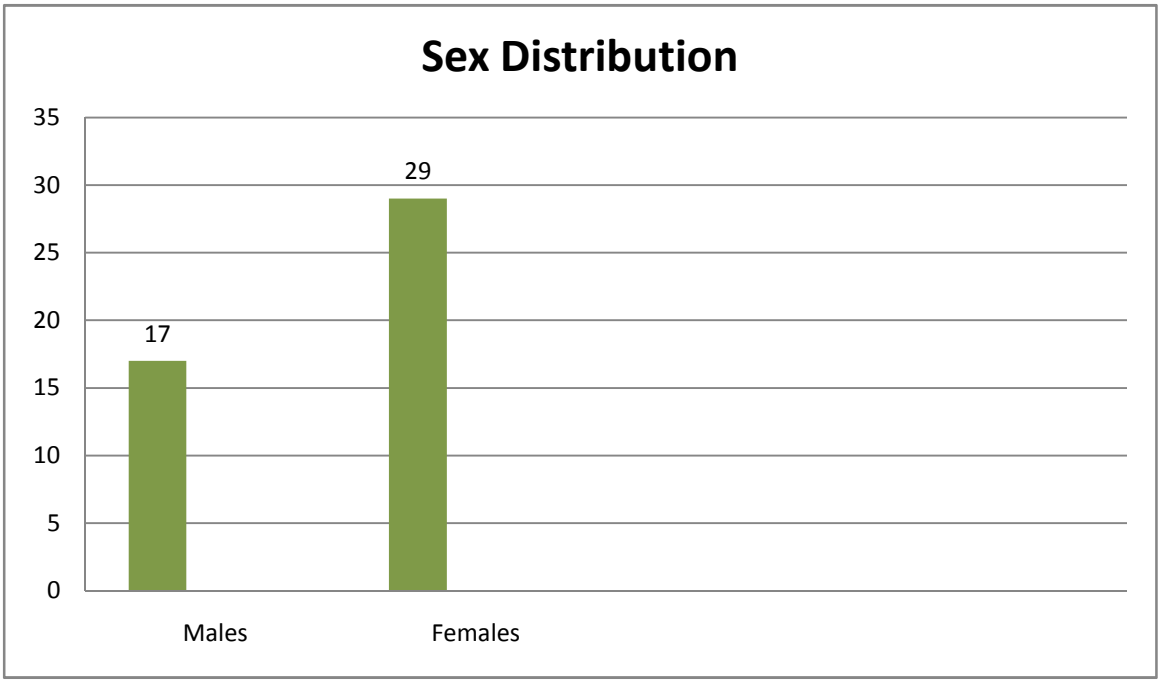

Fig.21

DVT in LSCS vs normal delivery

There was a higher proportion of cases of DVT in females who underwent caesarean section when compared to those who underwent normal delivery. This is understandably due to the prolonged period of immobilization and pregnancy itself which is a prothrombotic state. Out of the total of 26 pregnant females with DVT 18 had undergone caesarean section and 8 underwent normal delivery.

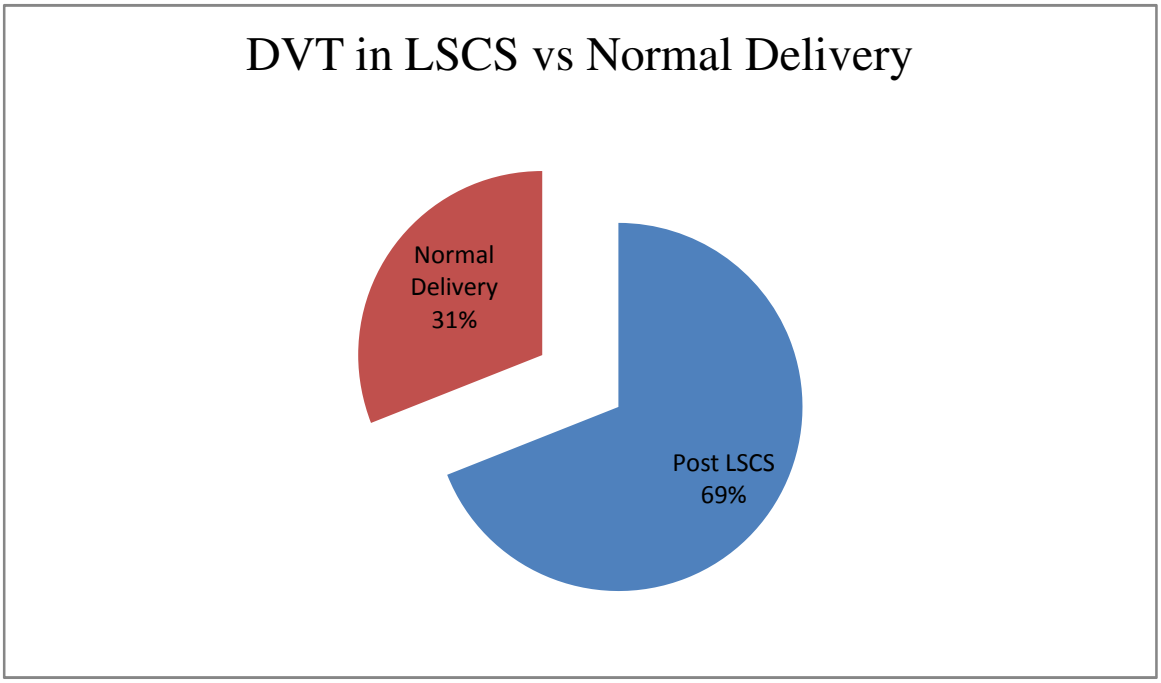

Fig.22

Age distribution of cases

The average age of male patients with DVT was 42 years whereas in case of pregnant females it was 27 years and non pregnant females it was 57 years.

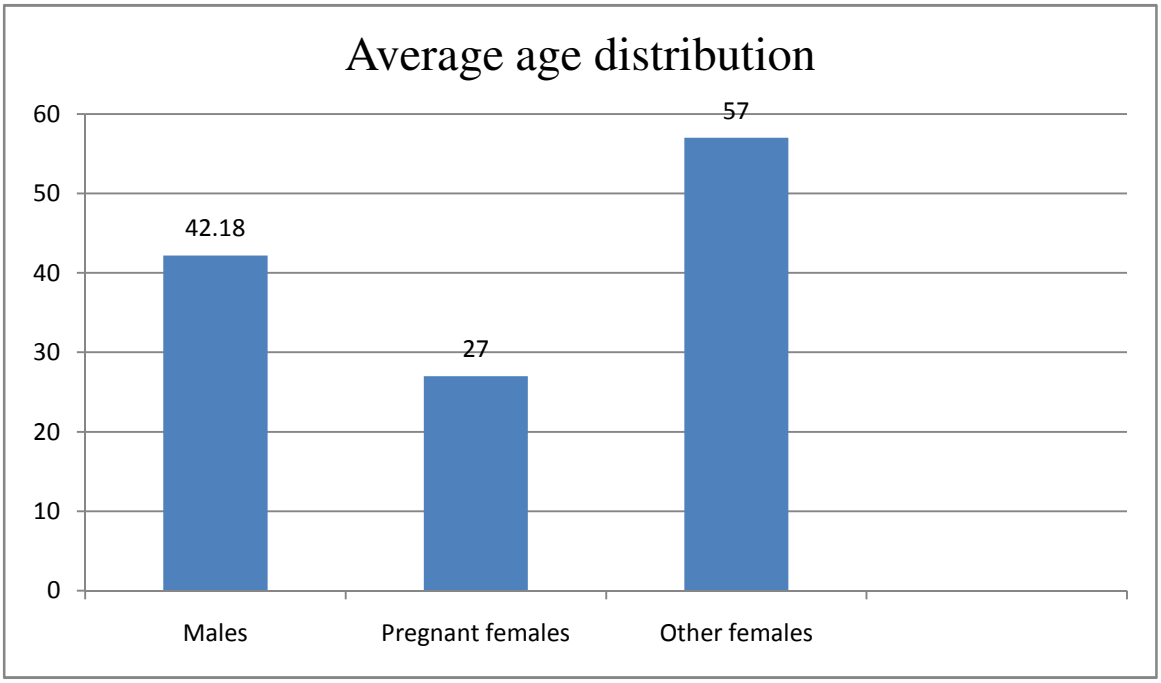

Fig.24

DVT in post op cases

Out of the total number of post op cases wit DVT 90% of the cases with DVT were post caesarean section followed by orthopaedics . No post operative cases in general surgery were reported to have DVT. This could be possibly due to early mobilization of the post operative cases and passive flexion exercises advocated in the post operative wards .

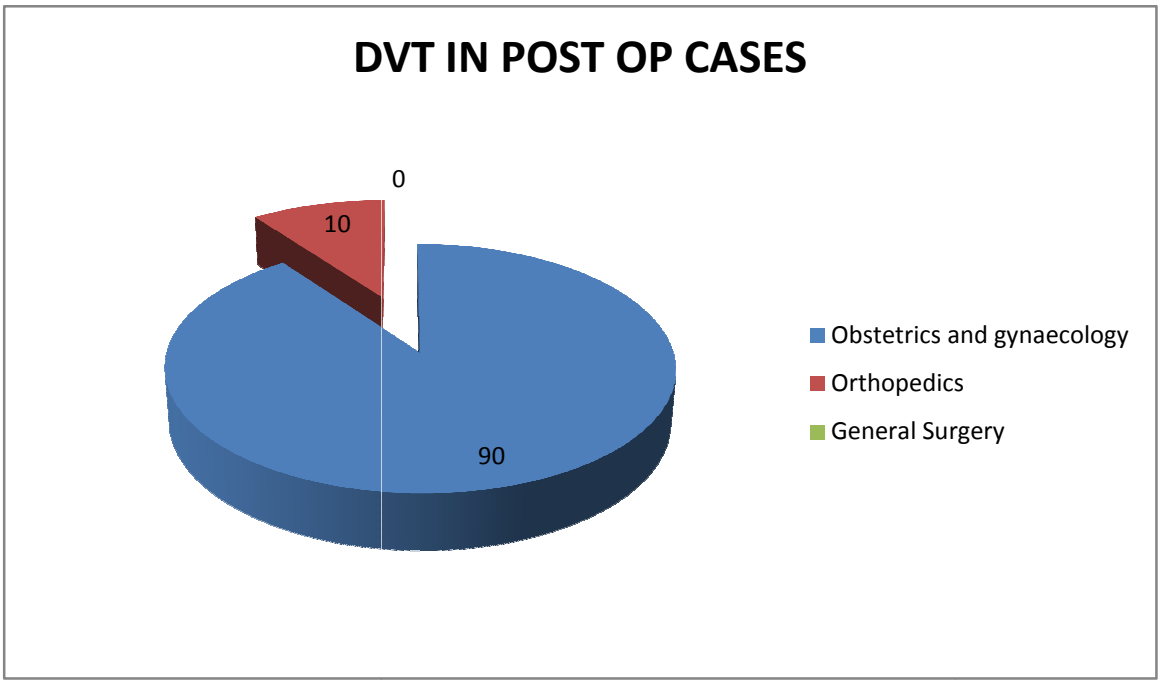

Fig.25

Total post op cases with DVT-20

LSCS cases -18

Orhtopedics cases- 2

General Surgery -0

Complications seen in the study group-

Out of the 46 cases of DVT 3 went in for complications. Out of the three cases 2 were female who had just delivered and had gone in for cerebral venous thrombosis. This was resolved by standard dose of heparin titrated according to

PT/INR values .One male patient died as he went in for cardiac complications due to myocardial infarction.

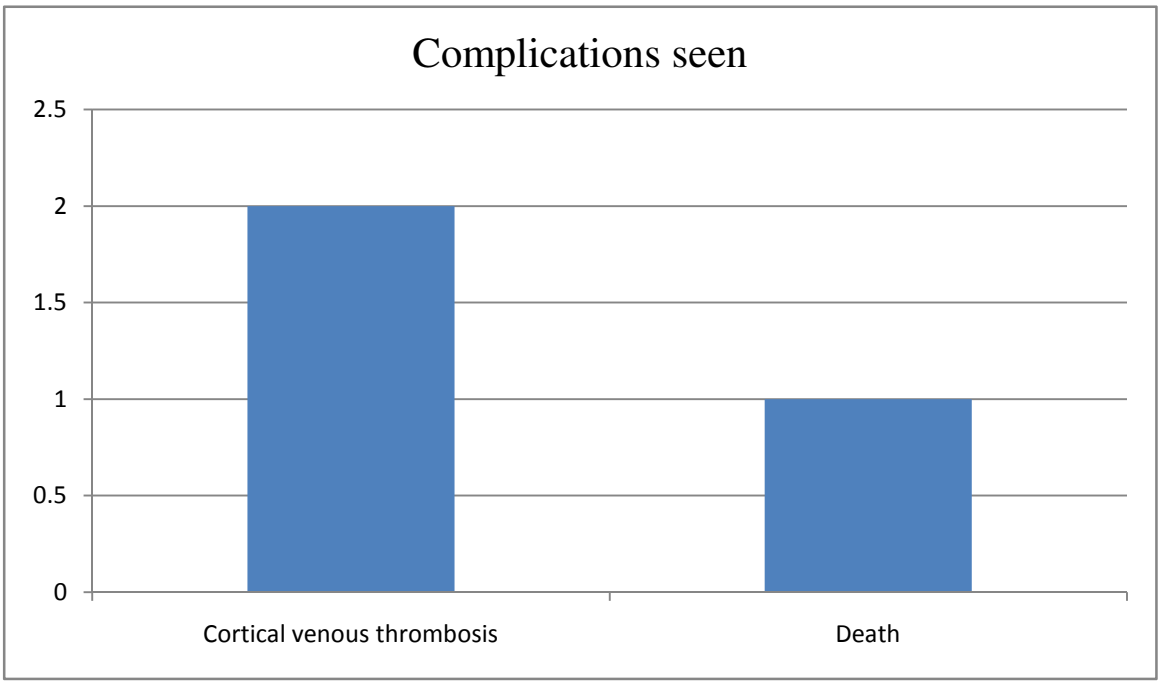

Fig.26

Analysis of co morbidities-

On analyzing the associated co morbidities in the study group it is clear that surgery was the most significant contributing factor resulting in DVT. In our study group most of the surgical cases were post caesarean section. Other co morbid conditions in descending order are smoking, dyslipidemia , obesity ,immobilization, cancer, myocardial infarction, renal failure and cancer. Finally a single case of IV drug abuse was also reported to have DVT.

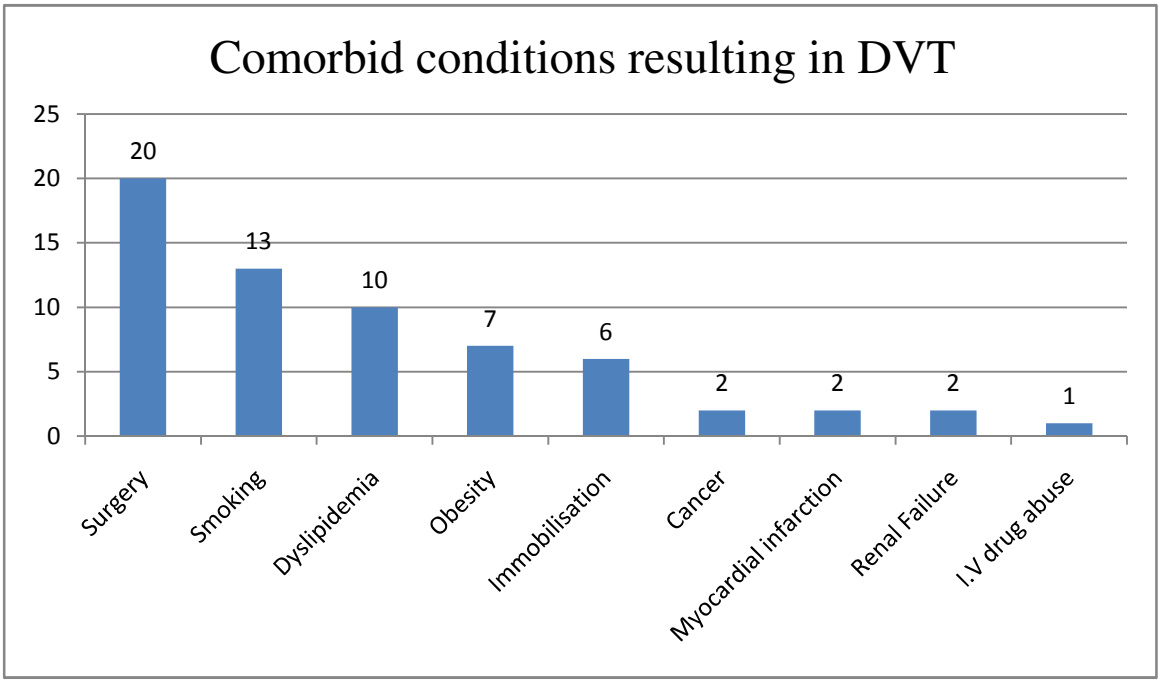
Comorbid conditions resulting in DVT
25
20
15
10
5
0
20
13
10
7
6
2
2
2
1
Surgery
Smoking
Dyslipidemia
Obesity
Immobilisation
Cancer
Myocardial infarction
Renal Failure
I.V drug abuse

Fig.27

DISCUSSION

Deep vein thrombosis is a condition which occurs in any location within the venous circulation. Majority of deep venous thrombi are known to occur within the deep veins of the leg , followed by the pelvis.

Occasionally deep vein thrombosis also occurs in the cerebral venous sinuses which was seen in two of the post partum cases in our study.

The Wells score was applied to 50 cases out of which 46 were proven to have deep venous thrombosis by Venous Doppler ultrasound.

Out of the proven cases of DVT there was a female predominance of cases in a ratio of 1.7:1 . Out of the total number of cases 63.04% was female and the remaining 36.96 were male

This is very much in concordance with a study done in New Zealand by Maelen Tagelagi and co-workers where 62% were women.[1]

However according to the New Zealand study the Wells Score was not so effective in their setup unlike the high predictive rate ie 46 out of 50 suspected cases seen in our study.

Among the false positive cases 2 were due to malignancy of the cervix who had underwent radiotherapy, the other two were due to early onset cellulitis.

Lymph edema is known to develop in cancer by various mechanisms. Extensive lymph nodal dissection following surgery is known to be the primary cause. Following surgery for cancer radiotherapy also causes scarring of the lymph nodes and lymph vessels.

Also tumor embolus is also known to block lymph vessels. Any cancer is a predisposing factor for unprovoked DVT which has been mentioned by Ruud Oudega and coworkers. [10]

Lymphedema also develops in cellulitis due to stagnation of protein rich lymphatic fluid which prove to be a rich medium for bacteria to grow thus creating a vicious cycle.

On analysis of the average Wells score seen in the DVT positive cases 29 out of the 46 cases had a Wells score of 4 out of 8 which specifies a 75% or greater chance of having DVT. [1]

The average age for male DVT cases in our study group was 42 years ,pregnant females 27 followed by a sudden rise in age which were mostly post menopausal women with cancer. Pregnant women have a five fold chance of having DVT compared to non pregnant women.[3,4,5]

This again correlates with the statistics given by Office of the Surgeon General (US) and the National Heart, Lung, and Blood Institute (US). [5]

It was also seen that there was a higher incidence of DVT in those individuals with non O blood groups. In our study majority of the cases of DVT were having a positive blood group thus indicating a higher level of plasma Von Willebrand factor, possibly a mutated variant. This correlated with the studies by Massimo Franchini and Mike Makris. [4,8]

Among the cases of DVT which had occurred in the post operative period, the highest incidence of DVT was following caesarean section, followed by orthopedics in polytrauma cases.

There was no case of DVT in the General surgery post operative ward in our study group. This could have been due to the early mobilization of cases in post op wards and advocation of passive flexion exercises.

There is also evidence in literature as written by Victor et al supporting that smoking also creates a procoagulant state by increasing platelet activation, decreasing fibrinolysis and various other mechanisms which puts smoking as a very high risk factor for DVT. [10]

Joel G Ray and co workers literature also suggests that dyslipidemia has a role to play in DVT which could also be seen in some of our cases. [9]

Finally there was a single case of IV drug abuse who had tested positive for DVT, the link between DVT and intravenous drug abuse being already proved by V A Cooke in England. [11]

CONCLUSION

- Among the 50 cases suspected DVT , the Wells score was able to predict DVT in 46 of the cases thus proving to be a very efficient diagnostic indicator.
- The average Wells score among the various cases was 4/8.
- Female cases of DVT outnumbered males in a ratio of 1.7:1.
- Considering the age distribution of cases males were in the 4^{th} decade followed by pregnant females in 2^{nd} decade and other females in the 5^{th} decade.
- The most important co morbid state for DVT is surgery especially caesarean section as seen in our study group as the patient is immobile as well as in a prothrombotic state.
- Among the cases of DVT post delivery, majority of them had developed DVT within the first 2 weeks following delivery whether via naturalis or LSCS, thus it is very important to promote early mobilization in the post partum period or even prophylactic anticoagulants with caution given the scale of the problem to decrease the morbidity in the post partum period.

- Complications noted in the study group were 2 cases of cortical vein thrombosis in the post partum period which fully recovered.
- Mortality rate in the study group was 4.3% in which a single case of diagnosed myocardial infarction died of heart failure.
- To sum up we can conclude that the Wells score is indeed a very good predictive criteria for deep venous thrombosis and can be applied with ease as it required only clinical assessment and thus avoids unnecessary delays in waiting for scans thereby allowing us to start anticoagulants as early as possible.

APPENDIX I

BIIBLIOGRAPHY:

1)Accuracy of the Wells Rule in diagnosing deep vein thrombosis in primary health care

Maelen Tagelagi, C Raina Elley

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3) Physiologic basis of Modern Surgical Care –Thomas A Miller

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7)Schwartz principles of Surgery -9th edition

8) Non-O blood group: an important genetic risk factor for venous thromboembolism

Massimo Franchini1 and Mike Makris

9)The role of dyslipidemia and statins in venous thromboembolism-Joel G Ray and Frits R Rosendaal

10) The Role of Smoking in Coagulation and Thromboembolism in Chronic Obstructive Pulmonary Disease-

Victor F. Tapson

11)Deep vein thrombosis among injecting drug users in Sheffield-V A Cooke and A K Fletcher

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16)Does This Patient Have Deep Vein Thrombosis?

Sonia S. Anand, MD et al

17) Lensing AWA, Prandoni P, Brandjes DPM, et al. Detection of deep-vein thrombosis by real-time B-mode ultrasonograph *N Engl J Med* 1989; 320: 342–45.

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Thromb Haemost 1991; 65: 233–36.

26) Importance of Pretest Probability Score and D-Dimer Assay Before Sonography for Lower Limb Deep Venous Thrombosis

Rathan M. Subramaniam[1 2 3], Tina Chou[1], Rebekah Heath[1] and Robin Allen[4]

27) Ganong's review of medical physiology

APPENDIX II

PROFORMA

Patient particulars

- Name
- Age
- Sex
- IP No
- Address
- DOA
- DOS
- DOD

History

- Complaints
- History of present illness
- History of Chronic Diseases

General Physical Examination

- Pulse
- Blood Pressure
- Temperature
- Hydration
- GCS

Examination of Abdomen

• Inspection

• Palpation

• Percussion

• Auscultation

• PR examination

Systemic Examination

• Respiratory System

• Cardiovascular System

• Central Nervous System

Investigations

• WBC Count

. Blood group

• Serum Sodium

• Serum Potassium

• Serum Bicarbonate

• Blood Urea

• Serum Cholesterol

• Chest X-Ray

• Ultrasound venous doppler

Wells Clinical Prediction Rule for Deep Venous Thrombosis (DVT)

Clinical feature	Points
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for more than 3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of greater than 3 cm (below tibial tuberosity)	1
Unilateral pitting edema	1
Collateral superficial veins	1
Alternative diagnosis as likely as or more likely than DVT	-2
Total points	

DVT = deep venous thrombosis.

Risk score interpretation (probability of DVT):

- /=3 points: high risk (75%);
- 1 to 2 points: moderate risk (17%);
- 1 point: low risk (3%).

APPENDIX III

MASTER CHART

s.no	Name	Age/sex	Ip.no	D.O.A	I.M	S.X	C.A	Smoking	M.I	R.F	T.A	B.G	I.V	O.B	DYS	PREG	W.S	DVT
1	Vellingiri	31/m	65215	6/10/12	No	No	No	Yes	No	No	No	A+	No	No	No	N.A	4/8	Yes
2	Ravi	45/m	66295	7/10/12	No	No	No	Yes	No	No	No	B+	No	No	Yes	N.A	3/8	Yes
3	Arumugam	33/m	67892	23/10/12	Yes	No	No	Yes	No	No	No	B+	No	No	No	N.A	4/8	Yes
4	Chandran	52/m	68253	2/11/12	Yes	no	No	Yes	No	No	No	AB+	No	No	No	N.A	2/8	Yes
5	Parvathy	70/f	70107	10/11/12	Yes	No	Yes	No	No	No	No	B+	No	No	No	N.A	5/8	Yes
6	Veluchamy	35/m	71225	14/11/12	No	No	No	No	No	No	No	A+	No	No	No	N.A	4/8	Yes
7	Chitra	35/f	72449	27/11/12	Yes	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
8	Janaki	38/f	75814	7/12/12	No	No	No	No	No	No	No	A+	No	No	No	N.A	4/8	Yes
9	Palaniammal	35/f	79854	15/12/12	No	No	No	No	No	No	No	B+	No	No	No	Yes	3/8	Yes
10	Mariyammal	26/f	78855	19/12/12	No	No	No	No	No	No	No	B+`	No	No	No	Yes	4/8	Yes
11	Janeth	27/f	923	1/1/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	3/8	Yes
12	Jamila	23/f	1021	17/1/13	No	No	No	No	No	No	No	B+	No	Yes	No	Yes	4/8	Yes
13	Arusamy	42/m	1572	18/1/13	No	No	No	Yes	Yes	No	No	AB+	No	No	Yes	N.A	2/8	Yes
14	Banupriya	21/f	1645	4/2/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
15	Shantha	45/f	2378	15/2/13	No	No	Yes	No	No	No	No	B+	No	No	No	N.A	5/8	Yes
16	Raman	47/m	3376	18/2/13	No	No	No	Yes	No	No	No	B+	No	No	Yes	N.A	3/8	Yes
17	Maragatham	41/f	4998	3/3/13	Yes	No	No	No	No	No	No	A+	No	No	No	N.A	3/8	Yes
18	Srinivasan	45/m	5279	15/3/13	No	No	No	Yes	No	No	No	A+	No	No	Yes	N.A	2/8	Yes
19	Jayalakshmi	23/f	5663	21/3/13	No	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
20	Easwari	25/f	6491	28/3/13	No	Yes	No	No	No	No	No	O+	No	No	No	Yes	4/8	Yes
21	Natarjan	52/m	7975	7/4/13	No	No	No	No	No	No	No	A+	No	No	Yes	N.A	3/8	No
22	Viatheeswaram	45/m	8331	9/4/13	No	No	No	No	No	No	No	A+	No	Yes	Yes	N.A	4/8	Yes
23	Lakshmi	21/f	9241	16/4/13	No	No	No	No	No	No	No	O+	No	No	No	Yes	3/8	No
24	Durgadevi	25/f	10214	29/4/13	No	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
25	Sumitra	30/f	15476	30/4/13	No	No	No	No	No	No	No	B+	No	Yes	No	Yes	4/8	Yes
26	Jayan	32/m	17582	2/5/13	Yes	Yes	No	Yes	No	No	Yes	A+	No	Yes	No	N.A	5/8	Yes
27	Nallammal	31/f	21884	11/5/13	No	No	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
28	Karthik	32/m	24662	12/5/13	No	No	No	Yes	No	Yes	No	B+	No	No	Yes	N.A	5/8	Yes
29	Perumal	45/m	27492	27/5/13	No	No	No	Yes	Yes	No	No	B+	No	No	Yes	N.A	4/8	Yes
30	Chinnammal	56/f	31226	5/6/13	No	No	Yes	No	No	No	No	B+	No	Yes	Yes	N.A	5/8	No
S.No	Name	Age/sex	Ip .no	D.O.A	I.M	S.X	C.A	Smoking	M.I	R.F	T.A	B.G	I.V	O.B	DYS	PREG	W.S	DVT
31	Dhandapani	54/m	34887	8/6/13	No	no	No	No	No	Yes	No	A+	No	No	Yes	N.A	4/8	Yes
32	Susheela	67/f	37922	9/6/13	Yes	No	Yes	No	No	No	No	A+	No	Yes	Yes	N.A	5/8	No
33	Ravishankar	45/m	38655	15/6/13	No	No	No	Yes	No	Yes	No	A+	No	No	Yes	N.A	4/8	Yes
34	Arumugam	70/m	39179	19/6/13	No	No	No	Yes	No	No	No	B+	No	No	Yes	N.A	4/8	Yes
35	Nataraj	55/m	47283	21/6/13	No	Yes	No	No	No	No	No	A+	No	No	No	N.A	4/8	Yes
36	Kannan	34/m	47349	26/6/13	No	No	No	Yes	No	No	Yes	B+	No	No	No	N.A	2/8	Yes

37	Rangammal	23/f	48927	4/7/13	No	Yes	No	No	No	No	No	B+	No	Yes	No	Yes	4/8	Yes
38	Darshini	27/f	50821	11/7/13	No	Yes	No	No	No	No	No	AB+	No	No	No	Yes	3/8	Yes
39	Shantalakshmi	25/f	51173	16/7/13	No	Yes	No	No	No	No	No	B+	No	No	No	Yes	4/8	Yes
39	Manoranjini	30/f	52746	7/8/13	No	No	No	No	No	No	No	O+	No	No	No	Yes	4/8	Yes
40	Sivakami	26/f	52739	12/8/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
41	Shanmugam	24/f	52882	18/8/13	No	No	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
42	Ranjini	28/f	54661	23/8/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	3/8	Yes
43	Easwari	29/f	55721	29/8/13	No	Yes	No	No	No	No	No	O+	No	No	No	Yes	3/8	Yes
44	Janaki	22/f	56982	1/9/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
45	Vanishree	26/f	61883	7/9/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
46	Lakshmi	25/f	62017	12/9/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
47	Raman	52/m	63715	23/9/13	No	No	No	No	Yes	No	No	B+	No	No	No	No	2/8	Yes
48	Subashree	31/f	63740	2/10/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes
49	Chinnal	28/f	66752	17/10/13	No	Yes	No	No	No	no	No	A+	no	No	No	Yes	3/8	Yes
50	Jayanthi	25/f	67931	19/10/13	No	Yes	No	No	No	No	No	A+	No	No	No	Yes	4/8	Yes

LEGENDS

DVT-deep vein thrombosis

D.O.A –date of admission

I.M-immobilization

S.X- Surgery done

C.A- cancer diagnosed case

M.I-myocardial infarction

R.F –renal failure

T.A –Trauma case

B.G –blood group

DYS-dyslipidemia

I.V –intravenous drug abuser

PREG- pregnant female

W.S-Wells Score

N.A- not applicable